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FOREWORD

The *United States Armed Forces Medical Journal* is the medium for disseminating information of administrative and professional interest to all medical personnel of the Department of Defense. The Chairman of the Armed Forces Medical Policy Council and the Surgeons General of the several services invite all medical officers, dental officers, Medical Service Corps officers, Nurse Corps officers, and officers of the Veterinary Corps of the Armed Forces, and the medical consultants of the Army, Navy, and Air Force to submit manuscripts for publication in this *Journal.*

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OFFICE OF THE SECRETARY OF DEFENSE
ARMED FORCES MEDICAL POLICY COUNCIL
WASHINGTON 25, D. C.

MEMO: Personnel of the Medical Services of the United States Armed Forces

It is with real regret that I find it necessary to resign my assignment in the Department of Defense in order to return to the private practice of surgery. It has been a privilege to serve, first as a member of the Armed Forces Medical Policy Council, and subsequently as its Chairman. I wish to express my deep appreciation to all the medical personnel who have co-operated with and contributed to the work of the Council.

The Secretary of Defense has made an excellent selection in his appointment of Dr. Melvin A. Casberg as the new Chairman. Dr. Casberg has been the Vice Chairman of the Council since 1 January of this year. He has had extensive experience in both military and civilian medicine and has served with the Army in the North African and the China, Burma, India Theaters during World War II, and as a surgical consultant to the Surgeon General of the Army since 1949.

Both to the new Chairman and to you, the personnel of the medical services, I wish the greatest success in the continued fulfillment of your mission.

W. Randolph Lovelace, II

W. Randolph Lovelace, II, M. D.

Wound Infection as a Complication of Ruptured Appendix[1]

Robert F. Christoph, *Commander, MC, U. S. N.*

Everett H. Dickinson, *Captain, MC, U. S. N.*

WITH the lowering of mortality in the presence of ruptured appendixes, our attention is now turned to a lowering of morbidity due to superficial wound infections which, from recent observations on the surgical service of this hospital, appears to be still too high In general, this occurred in patients in whom a perforated appendix with spillage and contamination of the peritoneal cavity was found at operation. In 55.5 percent of them a drain extending down to the peritoneum had been used and in 90 percent of these a persistently draining wound was the cause for continued hospitalization. The significance of this is further enhanced by the fact that in no case was there any intraperitoneal complication where the original infection had occurred. Excluding the average of 11 sick days for uncomplicated appendectomies, it was estimated that an average of 18 sick days per patient was caused by this complication alone. The purpose of this investigation was to ascertain by analysis of operative technic, pathologic findings, antibiotic therapy, and other factors whether this complication was avoidable in 36 patients admitted to this hospital from 1 April 1950 to 15 August 1951.

In a review of the recent literature one is impressed with the marked reduction of mortality following appendectomy. This reduction (1) began even in the presulfonamide era, probably because of better supportive treatment and education of laymen in the recognition of symptoms; (2) improved rapidly with sulfonamide therapy; and (3) continued to improve as other antibiotics have been added. Mueller *(2)* reported a decrease in mortality from 2.83 percent to 0.4 percent with the local

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(1) From the U. S. Naval Hospital, Oakland, Calif.

(2) Mueller, R. S.: Local use of sulfanilamide in treatment of acute appendicitis; review of 1,481 cases. Ann. Surg. 122: 625-630, Oct. 1945.

use of sulfanilamide and the intravenous use of sulfadiazine. Others reported still further decreases with the use of penicillin, streptomycin, and aureomycin (3-5). This reduction in mortality has been of prime importance but now more attention can be turned to wound infections. Few writers have included wound infection in their statistical analyses; none estimated it as a factor in increased morbidity; and few analyzed its causes.

CLINICAL MATERIAL

We have divided a series of 338 cases into simple (nonperforated) and perforated appendixes with a more detailed analysis of those that were perforated. The criteria of perforation were based on the surgeon's finding (1) a frankly perforated appendix with pus and occasional fecal soilage of the peritoneum, (2) a localized abscess, and (3) rupture on removal of the appendix. These were confirmed by the pathologists' reports. Although it is recognized that the appendix need not be ruptured or perforated to cause peritoneal infection and spreading peritonitis, we believed that it would be too difficult to correlate the interpretations of the various surgeons with reference to the amount or degree of peritonitis without perforation. This view is justified by Ravdin et al. (6). Of the 338 appendectomies reviewed in this report, 302 (89.3 percent) were classified as simple acute and 36 (10.6 percent) as perforated. About 81 percent of the patients were service personnel; the rest were dependents. The morbidity data of these groups were kept separate because dependents were often discharged to their homes and service personnel were hospitalized until fit for duty. A more complete study of morbidity was made with the latter group.

Duration of symptoms. The severity and extent of peritoneal soilage and peritonitis in our patients with perforation will not correspond with those of series from other clinics, especially charity hospitals where the patient often is not seen until he has suffered severely for 3 or 4 days and probably has taken a cathartic. The average duration of symptoms in the patients with perforation in our series was 53 hours (range, 8 hours to 13 days). This may be a factor in the low rate of intraperitoneal complications and in the absence of mortality.

Incision. Many writers stressed the importance of the McBurney incision as a factor in decreasing the mortality and morbidity with special reference to postoperative dehiscence and herniation. Meyer et al. (3),

(3) Meyer, K. A.; Requarth, W. H.; and Kozoll, D. D.: Progress in treatment of acute appendicitis. Am. J. Surg. 72: 830-840, Dec. 1946.

(4) Fauley, G. B.; Duggan, T. L.; Stormont, R. T.; and Pfeiffer, C. C.: Use of penicillin in treatment of peritonitis; experimental study. J. A. M. A. 126: 1132-1134, Dec. 30, 1944.

(5) Wright, L. T.; Schreiber, H.; Metzger, W. I.; and Parker, J. W.: Evaluation of aureomycin therapy in peritonitis; a report of 235 consecutive cases. Surg., Gynec. & Obst. 92: 661-671, June 1951.

(6) Ravdin, I. S.; Lockwood, J. S.; and Rhoads, J. E.: Use of sulfanilamide in treatment of peritonitis of appendiceal origin. Pennsylvania M. J. 43: 1100-1103, May 1940.

in a review of 5,543 cases of appendicitis, asserted: "The closer to the midline the incision is made, the higher the mortality rate." In our series 77.7 percent had McBurney incisions or minor modifications. The midline incisions were in women. Wound complications other than wound infection occurred in 0.9 percent.

Sutures. The use of cotton sutures, even in a grossly contaminated field that later broke down and drained, apparently had no ill effect per se. In no patient was the wound observed to extrude cotton sutures and once the drainage had subsided there were no recurrent flare-ups. The usual procedure was either closure of the peritoneum with running chromic No. 0 or 00 sutures and interrupted cotton No. 20 or 40 sutures to the fascial layers, or use of chromic No. 0 sutures on all layers. There was 90 percent wound infection in the incisions closed with chromic sutures and 46.1 percent in those in which cotton sutures were used.

Drains. This is a controversial subject and one which was investigated with some care, in the belief that it might have been a factor in the incidence of wound infections reported here. The use of intraperitoneal drains has been generally abandoned, except in patients having massive purulent abscesses with residual necrotic tissue left behind. Only 7 of these patients had this type of drainage. The use of drains extending down to the peritoneum is even more controversial. Because subcutaneous tissue has a low resistance to bacterial infection, many surgeons, even though courageous enough not to drain the peritoneal cavity, will insert a drain to the peritoneum or to the external oblique fascia. Several things have been noted about this type of drainage: The drains are usually removed by the second day and most wound infections were not sufficiently advanced to form exudate until the fourth day or later. Only 22.2 percent of the wounds closed without any drain had a wound infection, in contrast to 90 percent of those closed with a drain. The average time of onset of suppuration from wound infection was 6 days and most of the drains were removed after from 2 to 4 days. This leads to the conclusion that either the drains were not left in long enough or were not needed.

Bacterial cultures. The most frequent organism obtained on cultures of peritoneal fluid at the time of operation was *Escherichia coli.* Other bacteria commonly found were, in the order of frequency, *Aerobacter aerogenes, Proteus vulgaris,* aerobic nonhemolytic streptococci, paracolon bacillus, and *Pseudomonas aeruginosa.* This agrees with the studies of Harvey et al. (7) who concluded that there were always 2 or more varieties—*E. coli* always being present.

Antibiotics. It is difficult to get accurate clinical evaluations of the newer antibiotics. There is also some disagreement in the litera-

(7) Harvey, H. D.; Meleney, F. L.; and Rennie, J. W. R.: Peritonitis; Studies in peritoneal protection with particular reference to action of sulfonamide drugs in experimental peritonitis. Surgery 11: 244-260, Feb. 1942.

ture as to their intraperitoneal use. In this series 52.8 percent of the patients received a uniform course of penicillin and streptomycin (table 1). The average dose was 300,000 units of procaine penicillin twice daily and 0.5 gram of streptomycin every 6 hours. In some of the patients a quicker initial blood level of penicillin was obtained by giving 100,000 units of aqueous penicillin every 3 hours for 48 hours along with the procaine penicillin. High penicillin levels are needed in combating this type of infection. This has been brought out by Altemeier *(8)* who showed that a culture of *E. coli* destroys the activity of penicillin. Crile *(9)*, in reporting a similar series of cases, offered a similar theory for recommending massive doses of penicillin. He believed that *E. coli* in pure culture was not a pathogen and that large doses of penicillin were required to overcome the penicillin-destroying property of *E. coli* in order to obtain a level effective against the other organisms, Initially the rationale of penicillin therapy was put on a sound basis by Fauley et al. *(4)* who showed that large doses of penicillin in dogs will decrease mortality if given within 12 hours after perforation occurs.

TABLE 1. *Systemic antibiotics used in patients with perforated appendixes*

Antibiotic	Number of patients	Drain used	Wound infection	Combined wound infection and drain
Penicillin	3	2	3	2
Penicillin and streptomycin	19	15	14	14
Penicillin and aureomycin	9	5	7	4
Penicillin and chloramphenicol	1	0	0	0
Penicillin, streptomycin, and aureomycin	3	3	3	3
Total	35	25	27	23

Streptomycin, with its potent antibacterial action on a wide variety of both gram-negative and gram-positive organisms, especially those of gastrointestinal origin, makes it an effective agent in peritonitis. Its combination with penicillin is advantageous for several reasons. It exhibits its most important action on the penicillin-indifferent, gram-negative bacilli and gram-positive enterococci such as *Streptococcus faecalis*. Although many bacteria are susceptible to both streptomycin and penicillin, the range of sensitivity varies and penicillin-fast organisms may be susceptible to streptomycin and vice versa. Penicillin and streptomycin have a synergistic action against certain organisms

(8) Altemeier, W. A.: Inactivation of penicillin by various gram negative bacteria. Surg., Gynec. & Obst. 81: 379-383, Oct. 1945.

(9) Crile, G., Jr.: Peritonitis of appendiceal origin treated with massive doses of penicillin; report of 50 cases. Surg., Gynec. & Obst. 83: 150-162, Aug. 1946.

and certain combinations of organisms and multiple antibacterial agents decrease the ability of bacteria to develop resistance to them *(10, 11)*. Streptomycin is ineffective against *Clostridium welchii* and other anaerobic organisms and penicillin is moderately so, but the latter must not be discarded because Harvey et al. *(7)* have shown that *Cl. welchii* is frequently cultured from appendicial smears.

The two are compatible when both are used. Experimental peritonitis in dogs, reported by Murphy et al. *(12)* and Silvani et al. *(13)* indicated that the combination is more effective than or as effective as either one alone. Pulaski *(14)* reported that the combination gave the best results in peritonitis of intestinal origin, with streptomycin more effective in early-spreading peritonitis, and penicillin in the localizing infections.

Intraperitoneal use of antibiotics. It is difficult to evaluate the intraperitoneal use of antibiotics. We believe that the low incidence of intraperitoneal complications at this hospital is due in part to its use. Crile *(9)*, in his series of 50 patients, used sulfonamides intraperitoneally in only 5 and penicillin intraperitoneally in only 1; 7 of 24 operative patients developed intra-abdominal masses. In only 12 percent of our patients was even slight rectal tenderness or induration elicited. Avent et al. *(15)*, who did not favor the intraperitoneal use of antibiotics, reported only 1 death in 96 patients who were given penicillin and streptomycin intraperitoneally.

We believe that the rationale of the intraperitoneal use of antibiotics has been well established. The protective cellular responses of the peritoneum have been well described and utilized, Jackson and Coller *(16)* showed that 10 grams of sulfanilamide administered intraperitoneally produced a peak blood level of 10 mg. per 100 cc. in 2 or 3 hours with the local peritoneal concentration of the drug 100 or more times the level reached in the circulating blood. Mueller *(2)*, in a review of 1,451 patients treated locally with sulfonamides, showed a drop of mortality from 2.53 to 0.4 percent and a reduction in peritoneal

(10) Carpenter, C. M.; Bahn, J. M.; Ackerman, H.; and Stokinger, H. E.: Adaptability of gonoccocus to 4 bacteriostatic agents, sodium sulfathiazole, rivanol lactate, promin, and penicillin. Proc. Soc. Exper. Biol. & Med. 60: 168-171, Oct. 1945.

(11) Miller, C. P., and Foster, A. Z.: Studies on action of penicillin; therapeutic action of penicillin on experimental meningococcal infection in mice. Proc. Soc. Exper. Biol. & Med. 56: 166-169, June 1944.

(12) Murphy, J. J.; Ravdin, R. G.; and Zintel, H. A.: Use of streptomycin in experimental peritonitis. Surgery 20: 445-451, Oct. 1946.

(13) Silvani, H. L.; Rothenberg, S.; Warmer, H.; Amluxen, F.; and McCorkle, H. F.: Laboratory and clinical experiences with streptomycin therapy in management of infections of intestinal origin. Surg., Gynec. & Obst. 85: 721-726, Dec. 1947.

(14) Pulaski, E. J.: Use of streptomycin in treatment of surgical infections. In Andrus, W. DeW., et al. (editors): Advances in Surgery, Vol. II. Interscience Publishers, Inc., New York, N. Y., 1949. pp. 311-361.

(15) Avent, C. H.; Love, C.; Newman, R.; and Tendler, M. J.: Ruptured appendix; survey of 541 cases, 1939 through 1948. Surgery 27: 862-878, June 1950.

(16) Jackson, H. C., and Coller, F. A.: Use of sulfanilamide in peritoneum; experimental and clinical observations. J. A. M. A. 118: 194-200, Jan. 17, 1942.

abscesses from 2.5 to 1.3 percent. Shafiroff *(17)* showed that there was effective peritoneal absorption of penicillin. He gave 100,000 units of penicillin plus 100 cc. of plasma in normal peritoneums through a hernial sac. The highest blood level occurred 1 hour after instillation and effective levels were maintained for 5 hours. No untoward effects from penicillin given locally have been reported.

It has been shown that streptomycin must be used in maximal doses initially because organisms rapidly become resistant. Penicillin must also be present in heavy concentration where *E. coli* is present to overcome its inactivating factor *(9)*. Pulaski *(14)* stated that the peritoneal levels from intramuscular injections of streptomycin are one-half to one-fourth that of the serum level. Because penicillin diffuses rapidly into the peritoneal cavity *(11)*, it seems logical that the intraperitoneal use of streptomycin-penicillin solutions would quickly result in the desired concentration of both. No complications, such as adhesions, have been observed following this medication.

TABLE 2. *Intraperitoneal use of antibiotics in patients with perforated appendixes*

Antibiotic	Number of patients	Drain used	Wound infection	Combined wound infection and drain
Penicillin	8	8	7	7
Penicillin and streptomycin	14	12	11	10

It would seem from the results in our series that the patients who received the solution containing 1 gram of streptomycin and 600,000 units of penicillin had the smoothest postoperative course. Fourteen (39 percent) of these patients received streptomycin-penicillin intraperitoneally and 22 (62.5 percent) were given some antibiotic (table 2). We believe that this played an important part in the low incidence of complications and the smooth postoperative course.

Systemic use of antibiotics. From facts mentioned previously it was thought that penicillin-streptomycin therapy was the treatment of choice and most patients in the latter part of our series received it. The fact that the rate of excretion is slower with streptomycin than with penicillin permits giving the drug at less frequent intervals. Pulaski *(14)* suggested that the daily dose need not be more than 1.5 grams when given in combination with penicillin.

Mortality. There are still thousands of deaths from appendicitis each year. The Bureau of Census *(18)* reported 4,171 deaths in 1948,

(17) Shafiroff, B. G. P.: Peritoneal absorption of penicillin. Surgery 18: 753-754, Dec. 1945.

(18) Bureau of the Census, U. S. Department of Commerce: Statistical Abstract of the United States, 1950. p. 74.

a mortality rate of 2.9 per 100,000. In 1949 there were 8 deaths from this cause in all Navy and Marine personnel, a mortality rate of 0.2 percent (19). There were no deaths in the 338 patients reported here.

Morbidity. Attention was drawn to this aspect of appendicitis when 9 consecutive patients with perforated appendixes came to our ward postoperatively and each developed a wound infection. There were no other complications and clinically the patients were well enough for discharge on the tenth postoperative day except for their superficial wound infection (none extended through the peritoneum). Therefore, their average hospitalization period was 28 days. Appendectomies during previous years were then reviewed and the same fact stands out significantly. Here is a minor complication which added 18 days to the hospitalization of these patients. In our 36 patients, 26 developed wound infection. Despite the abundance of literature on appendicitis, very little has been written on this aspect. Many writers did not mention it; others included it without comment in tables of complications. Meyer et al. (3) stated that 120 of 592 patients treated with sulfonamides had purulent wound infections. Of these, 80 had no drain and 40 had drains extending to the peritoneum only

Medical officers in a military hospital are perhaps more conscious of this complication because the military patients are hospitalized until fit for duty. The average hospitalization of dependents for ruptured appendixes was 12 days, as compared to an average of 28.7 days for military personnel treated by the same surgical staff. Another factor is that most wound infections did not begin to drain until the fourth to tenth postoperative day. Many of these patients if they had been civilians could have been discharged to their homes before the infection was noticeable and treated subsequently on an outpatient basis. In 1949, 3,831 appendectomies were performed in the Navy with an average of 19.1 sick days (19). This included 66 patients whose diagnoses were changed to incision and drainage of appendicial abscesses and 5 whose diagnoses were changed to cellulitis and abscess, not elsewhere classified. Four of our 36 patients with perforation did not have the correct final diagnosis on the diagnostic summary sheet, being unchanged from appendicitis, acute, not elsewhere classified. This represents an error of 11.1 percent in this small series of cases reported.

Many factors enter into the cause of wound infections. Wound contamination, low resistance of the subcutaneous layer of fatty tissues, suture material, tissue strangulation, hematomas, drains, et cetera. The most important factors were wound contamination and hematomas or collection of serum. It was noted that laparotomy pads were infrequently used to protect the wound edges before opening the peri-

(19) Bureau of Medicine and Surgery, Navy Department, Washington, D. C.: Annual Report of the Surgeon General, U. S. Navy, to the Secretary of the Navy Relative to Statistics and Injuries in the United States Navy for the Calendar Year 1949. Navmed P-154. U. S. Government Printing Office, Washington, D. C., 1951.

toneum; that the appendix often brushed against the wound in the process of removal, and that the initial wound discharge was often serous and bloody, later becoming more frankly purulent, indicating that there might have been an accumulation of blood or serum which was a good medium for bacteria which otherwise could have been controlled by the local cellular response. Table 3 illustrates the relative importance of the superficial wound infections.

TABLE 3. *Complications*

In 36 patients with perforated appendixes	
Superficial wound infection	25
Induration and tenderness, rectal vault	4
Cardiac arrest with thoracotomy	1
In 269 patients with nonperforated appendixes	
Superficial wound infection	5
Atelectasis	3
Intra-abdominal abscess	2
Upper respiratory infection	2
Ileus, severe	2
Pyelitis	1
Meningism	1
Pelvic inflammatory disease	1
Salpingitis	1
Rectal mass	1
Glomerulonephritis	1
Pneumonitis	1
Duodenal ulcer	1

CONCLUSIONS

In a series of 338 patients with appendicitis there were no deaths. The rarity of intraperitoneal complications is believed to be due in great part to the intraperitoneal and systemic use of antibiotics. The superficial wound infections which added greatly to the morbidity, expecially of military personnel, was attributed mainly to the use of superficial drains and improper protection of the wound during operation.

An Outbreak of Q Fever

II. Treatment With Aureomycin and Chloramphenicol (1)

Francis X. Fellers, *Captain, U. S. A. F. (MC)*

PRIOR to the advent of aureomycin and chloramphenicol there had been no specific therapy for Q fever (2-5). Lennette et al. (6) reported success with aureomycin therapy in 1948. More recently chloramphenicol has been shown to be similarly effective (7).

Of a group of over 30 patients in Tripoli in whom Q fever was suspected, 25 were positively diagnosed as having this disease. The diagnosis was made by significant increases in the titer of the Q fever complement-fixation test on 22 patients; the other 3 patients were from the same living quarters and had a clinical course characteristic of the disease. Except for 1 outpatient included in this study, the patients were admitted to the station hospital over a 3-week period. Routine laboratory studies and roentgenograms of the chest were obtained prior to all specific treatment. Penicillin was given to the first 2 patients hospitalized. All received similar symptomatic and supportive therapy, including fluid replacement when necessary, antipyretics, and sedation. The 25 patients were separated into 4 groups as shown in tables 1 and 2. The first group contained 5 patients (including the nonhospitalized case and a patient with meningism). These patients had a mild febrile course with minimal toxicity. The average duration of the disease in this group was 5.1 days.

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(1) Part I was published in the United States Armed Forces Medical Journal, vol. 3, pp. 287-295, Feb. 1952.

(2) Huebner, R. J.; Jellison, W. L.; and Beck, M. D.: Q fever; review of current knowledge. Ann. Int. Med. 30: 495-509, 1949.

(3) Denlinger, R. B.: Clinical aspects of Q fever in southern California; study of 80 hospitalized cases. Ann. Int. Med. 30: 510-527, 1949.

(4) Oliphant, J. W.; Gordon, D. A.; Meis, A.; and Parker, R. R.: Q fever in laundry workers. Am. J. Hyg. 49: 76-82, 1949.

(5) Spicknall, C. G.: Terry, L. L.; and Huebner, R. J.: Treatment of Q fever with streptomycin; case report. Am J. Trop. Med. 28: 845-847, 1948.

(6) Lennette, E. H.; Meiklejohn, G.; and Thelen, H. M.: Treatment of Q fever in man with aureomycin. Ann. New York Acad. Sc. 51: 331-342, 1948.

(7) Zarafonetis, C. J. D., and Bates, R. C.: Q fever; report of case treated with chloromycetin. Ann. Int. Med. 32: 982-987, May 1950.

TABLE 1. *Control patients*

Patient	Duration of illness (days)	Admission temperature (° F.)	Roentgenogram of chest	Final Q fever complement fixation titer
Group 1				
4	6.5	101.2	+	1:160
6	7.2	99.0	+	1:40
8	6.2	99.2	+	1:20
22	2.5	100.6	+	1:40
28	3.0	-	-	1:80
Group 2				
2	10.5	100.2	+	1:160
5	8.5	102.2	+++	1:160
7	8.0	100.6	+	1:80
11	16.0	101.0	+	1:40
13	13.0	100.0	neg.	1:320
14	6.0	99.0	++	1:40
25	9.0	100.4	++	neg.

The other 20 patients had an abrupt onset of the febrile illness with constitutional symptoms and moderate to severe toxicity. These patients were ill on admission and were distributed as equally as possible in groups 2, 3, and 4. Group 2 consisted of 7 control patients who received no specific therapy; group 3, of 7 who received aureomycin; and group 4, of 6 who received chloramphenicol. The dosage schedules selected for therapy with these antibiotics were similar and consisted of a large initial dose followed by about 50 mg. per kilogram of body weight per 24 hours in divided doses for 5 days. Following the response of the first 4 patients treated, 2 with aureomycin and 2 with chloramphenicol, the dose was decreased to about 25 mg. per kilogram of body weight per 24 hours for the fourth and fifth days of treatment.

The aureomycin routine was as follows: A total dose of 11 or 9 grams of drug was given during a 5-day period; 250 mg. was given every hour for 4 doses. This was followed by 500 mg. every 6 hours until the course was completed. The schedule finally adopted substituted 250 mg. every 6 hours for the last 2 days of the course. The chloramphenicol routine was the same except that 500 mg. instead of 250 mg. was given every hour for the first 4 doses.

CASE REPORTS

Case 3. A 21-year-old airman was admitted to the hospital complaining of pain in the left side of the chest for several hours. For the previous 2 days he had noted a slight sore throat and a nonproductive cough. On the evening prior to admission he developed chills and fever, pain on breathing, and headache. On admission he appeared toxic and there was marked rigidity of the left side of his chest. His temperature

was 102.4° F. His pulse was 108. His respiratory rate was 20. There was moderate injection of the posterior wall of his pharynx and harsh breath sounds were heard in the left side of his chest posteriorly. The initial leukocyte count was 7,800 with 56 percent polymorphonuclear cells. Weil-Felix, heterophile antibody, and cold agglutinin reactions were normal. A roentgenogram of the chest showed a moderate-sized area of increased density in the midportion of the left lung which was interpreted as early consolidation at the base of the upper lobe (fig. 1). Pharyngeal culture gave many colonies of beta-hemolytic streptococci.

Treatment with penicillin in doses of 100,000 units every 3 hours was started on the day following admission. The symptoms and toxicity increased in the next 2 days. The fever persisted and the patient complained of stiffness in the neck and back. The neurologic findings were normal. The leukocyte count remained 7,700, but the roentgenogram of the chest showed extension of the areas of pneumonitis.

The patient was thought at this time to have an atypical pneumonia. The cold agglutinin titer was positive (1:80). The penicillin was discontinued after 48 hours and aureomycin was started with an initial dose of 1 gram followed by 10 grams in the following 5-day period.

Within 24 hours after starting the aureomycin therapy, the patient's temperature dropped rapidly to normal and he noted a dramatic relief of his malaise, headache, and chest pain. His toxicity vanished. A roentgenogram of his chest, taken on the second day of the aureomycin

TABLE 2. *Treated patients**

Patient	Duration of illness prior to treatment (days)	Admission temperature (° F.)	Roentgenogram of chest	Duration of fever after treatment (hours)	Total duration of illness (days)	Amount of antibiotic (grams)	Final Q fever complement fixation titer
Group 3 (treated with aureomycin)							
3	5.0	102.0	+++	30	6.0	11	1:160
9	5.0	102.2	++	36	6.5	11	1:40
12	12.0	100.0	+	28	13.0	9	1:40
17	5.0	103.6	neg.	24	6.0	9	1:160
20	5.5	102.0	neg.	24	6.5	9	1:80
24	7.5	100.8	+	24	8.5	9	1:40
26	4.5	101.0	+	52	6.5	9	1:40
Group 4 (treated with chloramphenicol)							
1	7.5	103.5	++	34	9.0	12	neg.
10	8.2	100.0	+	12	9.0	10	1:40
15	3.0	103.0	neg.	52	5.0	12	1:20
18	4.5	102.0	++	24	5.5	10	1:320
21	3.0	99.0	+	24	4.5	10	1:80
23	2.5	101.2	++	24	3.5	10	neg.

* The course of treatment was 5 days in each case.

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therapy, showed further extension of the pneumonic process. Subsequent films showed a gradual clearing. Therapy was discontinued after 3½ days of normal temperature. The patient was discharged to duty 2 days later and followed in the outpatient clinic. The cold agglutinin titer on the twelfth day after the onset was 1:40. By this time acute Q fever was suspected. Confirmation of the diagnosis was received 2 weeks later from complement-fixation studies. The titers for this patient were as follows:

Days after onset	*Q fever titer*
6	Anticomplementary
15	1:40
50	1:160

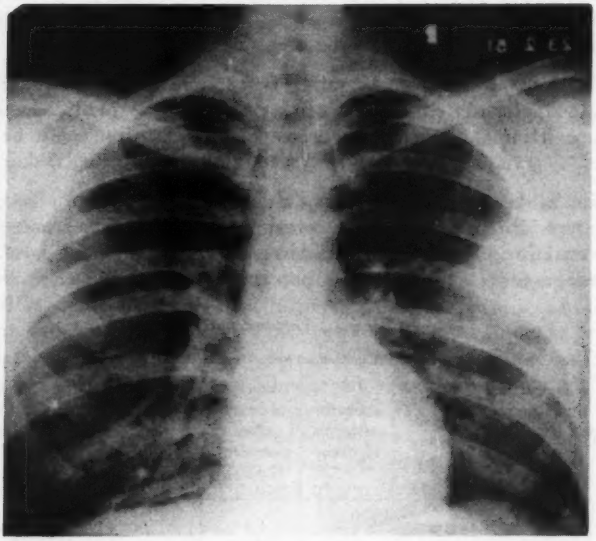

Figure 1.

Case 10. A 20-year-old airman was admitted to the hospital complaining of chills and fever of 12 hours' duration. During the 2 preceding days he had headache, slight sore throat, and backache. On the day of admission he developed a nonproductive cough. On admission he appeared ill. His temperature was 100° F. His pulse was 80. His respiratory rate was 20. The only positive findings on the physical examination were some injection of his pharynx and exudate in the crypts of his right tonsil. His leukocyte count was 8,400 with 71 percent polymorphonuclear cells. Weil-Felix, heterophile antibody, and cold agglutinin reactions were normal. Pharyngeal culture gave a moderate number of beta-hemolytic streptococcal colonies. A roentgenogram of

the chest revealed a discrete area of density in the midportion of the right lung. A first strength tuberculin test was negative at 48 hours.

After the first hospital day the patient felt better, but his temperature continued to rise gradually and he had occasional chills. By the third hospital day his temperature reached 103° F. Chloramphenicol therapy was started and he received the 10-gram course in a 5-day period. A prompt response was apparent in 12 hours with a drop in his temperature. Within 36 hours he volunteered that he felt fine. At the end of the therapy a roentgenogram of the chest showed some decrease in the size of the pneumonic area. He was discharged to duty 2 days later. Follow-up studies confirmed the diagnosis of Q fever in this patient as follows:

Days after onset	*Q fever titer*
5	Negative
23	Negative
49	1:40

RESULTS

The results are summarized in table 2. The response was dramatic with either aureomycin or chloramphenicol. A marked subjective improvement was noted by the treated patients about 12 hours after starting specific therapy. On an average, within 30 hours after starting treatment, the patient's temperature had dropped to normal and the headache, malaise, and photophobia had disappeared. The natural course of the disease, as determined in the control group of 7 patients not treated with antibiotics, showed an average duration of febrile illness of 10.1 days. The patients in the aureomycin-treated group were ill an average of 6.4 days prior to therapy. This group showed a return to normal temperature 31 hours following the oral administration of aureomycin. The patients in the chloramphenicol-treated group were ill 4.8 days prior to specific treatment. Following therapy the temperature returned to normal in an average of 28 hours.

The response to aureomycin therapy was observed in 2 patients who had a persistent headache following the initial febrile stage of the disease. Both of these patients had a mild infection and had not been considered for therapy at the onset. Patient 4 was in the hospital for 2 weeks following the acute phase of his illness with a persistent headache and negative physical findings. On his twentieth day treatment with 1 gram of aureomycin daily was started. This was continued for 3 days. During this treatment his headache gradually improved and he was discharged to duty 2 days later. The other patient (case 6) had the symptoms and signs of a meningism. His spinal fluid was completely normal. His headache persisted and, consequently, on the eighteenth day of his illness, treatment with 1 gram of aureomycin daily was started. This was continued for 4 days. There was no change in his

headache. He was then discharged to duty and follow-up revealed that the headache had gradually cleared during the week following discharge.

No significant changes were observed in the laboratory studies, including white blood cell counts, differential counts, and the Weil-Felix, heterophile antibody, and cold agglutinin reactions. Both treated and nontreated patients developed complement-fixing antibodies for Q fever. Of the 2 patients who did not develop antibodies by the twelfth week, 1 was in the control group and 1 was in the chloramphenicol-treated group. Another of the chloramphenicol-treated patients could not be reached for the follow-up studies. Side effects were minimal with both of the antibiotics used. One patient experienced nausea, and vomited once, while taking the aureomycin capsules. Two patients complained of poor appetite without nausea while taking chloramphenicol.

DISCUSSION

Aureomycin previously had been employed successfully in Q fever *(6,8)*. The dosage used by these authors ranged from 2 to 5 grams of aureomycin daily, with variable duration, generally from 1 to 2 days after the temperature had returned to normal. Reports on the treatment of older patients with aureomycin described the temperature as returning to normal on an average of 3 days after starting therapy and symptomatic improvement within 48 hours. In one patient *(8)* 3 grams of aureomycin were given daily for 4 days. The temperature returned to normal in 2 days. Relapses were reported in some of these patients.

The results in the present series of 7 patients treated during the acute phase of Q fever with aureomycin again showed the dramatic response of this disease to aureomycin. The average duration of the untreated hospitalized patient with this disease has been reported as 10 days *(3)*. This corresponds closely with the duration of febrile illness (10.1 days) found in the present untreated control group. Thus aureomycin appreciably shortened the febrile period by 2.5 days in this series. In the 2 patients who were treated with aureomycin after the acute phase for the headache associated with Q fever, the results were inconclusive. A greater dosage might have given a specific therapeutic response as Lennette et al. *(6)* showed that a longer course of therapy was necessary when the disease had persisted for many days. The final dosage schedule of 9 grams of aureomycin in a 5-day period is suggested as adequate therapy for patients with acute Q fever.

Chloramphenicol has also been successfully used in the treatment of Q fever, but few cases have been reported. Harmon *(9)* reported a patient with this disease who was given 6.25 grams of chloramphenicol during a 36-hour period. Treatment was begun on the seventh day of the

(8) Waldo, H. H.: Q fever. U. S. Armed Forces M. J. 2: 921-926, June 1951.

(9) Harmon, J. B.: Treatment in Q fever with chloromycetin; case report. Lancet 2: 1028, 1949.

febrile illness. Within 24 hours the temperature became normal and the symptoms disappeared in the following 24 hours. Zarafonetis and Bates *(7)* reported a patient with Q fever who was treated with chloramphenicol. He received a total of 22.5 grams of the drug over a 9-day period, beginning on the fourth day of his illness. Improvement began in 18 hours and his temperature became normal in 48 hours. The results in the present series of 6 patients treated with chloramphenicol showed a similar dramatic response. Treatment with this drug decreased the duration of fever by 4.1 days. Zarafonetis and Bates, using the experimental data of Smadel and his associates, suggested that Q fever might require a relatively greater dosage of chloramphenicol than is given for similar rickettsial diseases. Because one probable relapse occurred in this series treated with chloramphenicol, 10 grams of this drug in a 5-day period is suggested as a minimal therapeutic regimen for acute Q fever.

A comparison of these 2 antibiotics as used in this study showed no appreciable difference in the early therapeutic response. Further study is needed to determine whether Q fever would respond to larger doses of specific antibiotics given in the first 24 to 36 hours of treatment as described recently for other rickettsial diseases *(10)*.

CONCLUSION

In a study of 25 patients with Q fever, both aureomycin and chloramphenicol were found to produce a dramatic therapeutic response.

(10) Woodward, T. E.: Chloromycetin and aureomycin; therapeutic results. Ann. Int. Med. 31: 53-82, July 1949.

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The Use and Misuse of Radiologic Terminology

Peter Zanca, *Colonel, MC, U. S. A.* (1)

WITH the development of the science of radiology a descriptive vocabulary came into existence. The correct usage of this terminology conveys an exact and specific meaning. The proper interpretation of the normal and abnormal physiologic processes of the body, from the study of the roentgenologic findings, expressed in radiologic language is an established procedure and is frequently taken for granted by the specialist and general practitioner.

Although every physician has a variable and extensive medical vocabulary, his acquired radiologic terminology varies in proportion to his training and experience in the field of radiology. The necessity of bringing to the attention of the medical profession the changes that are constantly being made in the growing radiologic terminology and the need for re-emphasizing some of the more practical uses and misuses of this language become more and more evident. Corrective measures should make for better understanding between the radiologic service and the other hospital services and should help to establish that confidence in the professional team which aims to render the best medical care for the patient.

EXAMPLES

Inaccurate use of anatomic terms.

1. Requests for "x-ray of the hand to rule out fracture of the carpal bones" are often received. Such requests should read: "X-ray of the wrist———." The part of the body to be x-rayed should be described accurately because usually only the views taken of that region will show the lesion present. In this case, the roentgenograms taken in ulnar or radial deviation might reveal fractures which could have been missed on the routine roentgenogram of the hand.

2. Requests for "x-ray of the arm" may be received unaccompanied by any other information. Inquiry later may reveal that the physician

(1) U. S. Army Hospital, Camp Cooke, Calif.

wanted to rule out a Colles' fracture. The request should read: "X-ray of the forearm." The x-ray technician under such circumstances would have roentgenographed the wrong part of the body.

3. Similarly, requests for "x-ray of the leg" are received when the physician ordering the film wanted to rule out fracture of the femur and should, therefore, have requested an "x-ray of the thigh."

4. Requests for "x-ray of the back" in patients who complain of pain in the back are often received. In such cases, the part of the back involved should be specified. Thus, if the suspected lesion was in the "small of the back" roentgenograms of the lumbosacral region should be ordered.

Inaccurate usage and interpretation of descriptive terms. Requests for a "lateral view of the chest" with no other information may be received. Inquiry later may reveal that the referring physician wished to rule out the presence of free fluid in the pleural cavity. In this case the request should read: "Right (or left) lateral decubitus film of the chest." Like the upright chest film, the lateral decubitus film would demonstrate the presence or absence of a fluid level, whereas a lateral view of the chest, taken from above with patient in recumbent position, would not demonstrate a fluid level.

Use of obsolete phrases.

1. Requests for a "flat plate of the abdomen" hark back to the days the roentgenogram was made on a glass plate. The request, in such cases, should read: "Plain film of the abdomen."

2. Requests for a "Graham-Cole test" are likewise obsolete. The roentgenologic examination of the gallbladder is routinely being done with the use of iodoalphionic acid. The Graham-Cole test refers to an examination formerly performed after the administration of tetraiodophenolphthalein. Such requests should be made for a "cholecystographic examination" or for a "gallbladder series."

Misapplication of terms.

1. Requests for "G. I. analysis" are misleading in that the gastric analysis is performed by the laboratory service and not by the x-ray service. If a study of the upper gastrointestinal tract, by the fluoroscopic and roentgenographic methods, is desired the request should be made for a "G. I. series."

2. In requesting that a roentgenogram be taken, the expression "take a picture" is commonly used. In photography, "taking a picture" refers to making an exposure on negative material. It would be better for the request to read: "Have a roentgenogram of———taken."

Use of vague, inaccurate, nonscientific words and phrases.

1. Requests for a "lower G. I. series" are subject to misinterpretation. Is the request made for a barium-meal study of the small intestines

or does the physician desire to rule out a lesion in the large bowel? The request should specify the part of the gastrointestinal tract to be studied. For the small intestines, a motor-meal examination is a good procedure. For the large bowel, the routine barium enema examination or the double-contrast air study should be requested.

2. A request is sometimes made to have an "x-ray film taken in an inclining position." The correct descriptive term for the position desired is the "recumbent (or reclining) position."

3. Request may even be received for an "x-ray of the patient on his stomach (or on his belly)." The more acceptable terminology, in this case, would be to "have the patient in the prone position."

Redundancy. In the expression "roentgen units" the word "unit" is redundant because the roentgen, per se, is a unit and represents a specific number of ion pairs per unit mass.

The correct interpretation of radiologic reporting. In reporting the "5-hour roentgenogram" of a gastrointestinal series examination, the radiologist frequently incorporates the following statement in his report: "At 5 hours the head of the barium column is in the splenic flexure and the tail of the barium meal is in the ascending colon." The description used in this case is a good example of radiologic reporting of what is seen on the roentgenogram. The radiologist may not explain the clinical significance of this finding, but the implication is that there is normal progress of the barium meal which suggests normal function of the gastrointestinal tract examined.

Miscellaneous.

1. A request may be received for a "routine x-ray of the chest." Inquiry later may reveal that the roentgenogram is to be used for measurement of heart size. In some x-ray departments the routine roentgenogram of the chest is taken at a distance of 48 inches. This roentgenogram would not be satisfactory for accurate measurement of heart size. The request should be made for a "teleroentgenogram of the chest."

2. A request for "conventional views" is in good form because all of the routine exposures are implied. In the case of the skull 5 routine exposures are made. The routine roentgenologic procedures vary from hospital to hospital only in minor details and according to the facilities and equipment available. Every x-ray department, however, has a list of standard procedures which have been adopted to conform with accepted standards and which serve to satisfy the local needs of the various specialties.

DISCUSSION

Corrective measures and supervision of requests for roentgenologic procedures are functions of the radiologic service. In institutions

where there is a constant turnover of personnel, the problem of teaching becomes time consuming. It is believed, therefore, that it would be most profitable if the physician were to inform himself of the locally established routine procedures and then to request the more specialized procedures after consultation with the radiologist. Some of the newer and more commonly used specialized roentgenologic procedures and the terminology applied are: kymography, electrokymography, orthodiagraphy, laminography, aortography, arteriography and venography, angiocardiography, arthrography, scanography, and cerebral angiography.

LOST HISTORICAL DOCUMENT

A very important Army Medical Service historical document is missing from the Army Medical Service Graduate School. It is a red, ledger-type book, measuring about 9 by 14 inches, labeled "Records of the Army Medical School, Vol. I, Session 1 to 9, June 1893 to June 1904." The first page of this book contains a copy of General Orders No. 51, Headquarters of the Army, June 24, 1893, establishing the Army Medical School. This is followed by longhand recording of the minutes of the first faculty meeting, signed by Walter Reed.

Anyone having information which might lead to the recovery of this book, please contact Colonel William S. Stone, MC, U. S. A., Commandant, Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington 12, D. C.

Smallpox

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Robert C. Ziss, *Lieutenant, junior grade, MC, U. S. N. R.* (2)

SINCE the introduction of cowpox vaccination, the incidence of smallpox in the Western world has steadily declined to such low levels that its prevalence in Oriental countries is frequently forgotten. Its presence and virulence in Asia was forcibly impressed on medical officers in Korea and Japan during the winter of 1945-46 when 121 cases of smallpox with 25 deaths occurred in presumably immunized military personnel (3). In view of present far-flung military activities, particularly in Korea, further encounters with this disease are to be expected.

The present report deals with 2 cases of smallpox in American personnel, observed and treated aboard a Navy hospital ship operating in Korean waters. Both patients had recently been vaccinated more than once, but no history of a "take" could be elicited. That these vaccinations conferred no immunity was verified in each instance by the severity of the attack. In addition to supportive therapy, one patient received penicillin and aureomycin and the other was given a combination of penicillin and chloramphenicol. Although both patients recovered, in neither case did the natural course of the disease appear to be altered by this therapy.

CASE REPORTS

Case 1. A 21-year-old man was admitted to the U. S. Naval Hospital aboard the U. S. S. *Consolation* on 29 October 1950. He had been well until 3 days before admission when he was suddenly beset with fever, vomiting, abdominal pain, headache, and backache. Most distressing was the protracted vomiting, which made even the retention of small amounts of fluid impossible. The fever ranged from 102° to 104° F. each day and bilateral lumbar pain was especially prominent.

The patient had served with a Marine Corps unit in Korea until 12 days before his illness when he boarded a transport. He denied any

(1) U. S. Naval Hospital, Chelsea, Mass.

(2) U. S. S. *Fremont.*

(3) Smallpox in Japan and Korea. (News and Comment section.) Bull. U. S. Army M. Dept. 6: 101-102, Aug. 1946.

contact with a known case of smallpox aboard the ship. He had been vaccinated at the age of 12 years, again on entry into the service in 1948, and 4 times in August 1950. Vaccinia did not follow any of these attempts.

On physical examination he was found to be acutely ill. His temperature was 103° F. His pulse was 92. His respirations were 28. His tongue was thickly coated. His throat was markedly injected. There were 2 subconjunctival hemorrhages in his right eye. His neck could not be fully flexed. There was moderate tenderness over his abdomen and lumbar musculature. No rash, vaccination scar, or petechias were present. The urine contained a trace of albumin and the specific gravity was 1.035.

Intravenous fluids were administered and 300,000 units of penicillin were given once daily. On the following morning the patient appeared near prostration. The fever and all other symptoms persisted. In addition, for the first time a red maculopapular eruption consisting of distinct lesions measuring from 2 to 5 mm. in diameter was noted. This was concentrated primarily over the face and torso, with some lesions scattered over the extremities. The hands and feet were not involved.

On the next day the rash was more widespread and a few lesions were topped with vesicles. Aureomycin therapy in doses of 500 mg. every 6 hours was started. Within another 48 hours, vesiculation of the rash was complete. The face, scalp, and body were most heavily involved and the pharynx also was studded with vesicles. A few vesicles appeared on the hands and feet, including the palms and soles, but the involvement in these areas was far less than that on the face and body. The temperature hovered about 101° F. but the patient felt somewhat improved and his vomiting ceased.

In the ensuing days, the vesicles, many of which were umbilicated, became turbid and took on a dirty yellow hue, which contrasted markedly with the erythematous base on which they sat. On the twelfth day of the disease (eighth day of the rash) the patient presented the classical pustular eruption of smallpox (fig. 1). The face was markedly swollen and the cervical lymph nodes were large and tender. The temperature which had gradually fallen to normal rose to 101° F., but the patient complained only of pain on swallowing and severe pruritus. The latter was adequately controlled with benadryl and Burow's solution soaks. The leukocyte count reached a peak of 13,600 at this time. The differential count remained within normal limits. The albumin disappeared from the urine. There was no growth from a blood culture taken on admission.

Rapidly thereafter the face became crusted but elsewhere the eruption flattened and turned a dark hemorrhagic red. The resultant scabs and crusts gradually fell away. The leukocyte count fell to normal levels but the percent of lymphocytes and eosinophils increased to 58

and 16 percent respectively. On the twenty-second day of the disease the skin was clear. Following a 2 weeks' convalescence the patient was able to return to duty. At the time of discharge an ECG was normal. The face showed a moderate amount of pitting, but elsewhere there was no scarring.

Case 2. A 17-year-old seaman was admitted to the U. S. Naval Hospital aboard the U. S. S. *Consolation* on 27 January 1951 complaining of aching back and leg muscles, a sore throat, headache, and a severe nonproductive cough of 2 days' duration. He had stabbing pains in the right lower abdominal quadrant and frequent vomiting. Hyperpyrexia, up to 105° F., associated with a marked progressive prostration developed. A lumbar puncture performed before admission revealed a normal fluid under normal tension. Treatment before entrance into the hospital consisted of penicillin and intravenous fluids. The patient had set foot in Korea for the first time just 13 days before the onset of his illness. About 1 week prior to that he had been in Japan, but previously had never been out of the United States. He had been vaccinated 1 and 6 months before his illness, but neither vaccination "took."

Physical examination revealed an acutely ill young man with a temperature of 104° F. His nasopharynx was markedly injected and dehydrated. A yellow exudate covered his posterior pharyngeal wall and his left tonsil. There was moderate tenderness to deep percussion over both kidneys. A roentgenogram of the chest showed a small area of pneumonitis in the lower part of the left lung.

Fluids were given intravenously and treatment with 100,000 units of penicillin every 4 hours was started. On the second hospital day a red maculopapular rash which consisted of firm separated punctate lesions began to appear over the entire body. The sites of predilection included the face, soft palate, arms, and legs. On the following day a few papules became vesicular and no area of skin or visible mucosa was without lesions, but a centrifugal tendency was obvious. With the full flowering of the rash, the patient improved subjectively. His temperature fell to 99° F. and he was able to retain food for the first time. At this stage treatment with 500 mg. of chloramphenicol every 6 hours was begun. The penicillin schedule was changed to a single injection of 600,000 units daily. Burow's solution soaks were started and these were continued throughout the course of the disease.

On the sixth day of the disease the vesicular stage was evident over most of the body. On the next day pustules appeared on the face and the process gradually extended downward to be complete on the tenth day (fig. 2). The temperature rose to 102° F. and the patient became extremely uncomfortable. There was marked facial edema and the itching of the pustules was intense. The lesions in the mouth and throat made swallowing painful and a liquid diet became necessary. Barbiturates and pyribenzamine were used to allay the discomfort. The

Figure 1 (case 1). Appearance of pustules on twelfth day of illness (eighth day of the rash). Figure 2 (case 2). Appearance of pustules on tenth day of illness (seventh day of rash).

leukocyte count, which was 6,900 initially, varied between 10,000 and 11,500 during the pustular stage. A throat smear taken on admission grew beta-hemolytic streptococci on culture. A blood culture taken at the same time showed no growth. A roentgenogram of the chest revealed complete disappearance of the pneumonitis originally present.

Crusting, which began on the face on the eleventh day, was not generalized until the sixteenth day and it was not until the fortieth day of the illness that the last crust dropped off and the patient was removed from isolation. Shortly after the onset of crusting the leukocyte count fell to normal, but the lymphocytes which accounted for only 16 percent of the differential count in the beginning rose to 57 percent and the eosinophils reached 7 percent. Starch baths and lanolin applications were used to soften the lesions in the terminal phase in an endeavor to minimize scarring. During his illness the patient lost 30 lb., but his appetite and sense of well-being returned sufficiently for his transfer to a hospital in Japan at the end of 8 weeks of hospitalization. An ECG taken just before transfer showed no evidence of cardiac damage. The skin was severely pitted, but because epithelial regeneration was not yet complete, the final outcome could not be predicted.

DISCUSSION

The clinical diagnosis of variola depends on the prodromal symptoms and the rash. Before the appearance of the rash, the disease cannot be distinguished from a host of other acute infections. In the cases presented, the initial diagnosis in each instance was meningitis. Once the rash appears, the diagnosis becomes obvious in unmodified cases if one is at all alert to the possibility of smallpox. In modified cases or those in which the eruption is hemorrhagic, the diagnosis may remain obscure. The modified cases can be exceedingly difficult to differentiate from chickenpox and, according to Illingworth and Oliver *(4)*, an early diagnosis between the two is often impossible without recourse to laboratory study. These observers also drew attention to the fact that in all of 78 nonhemorrhagic cases the specific rash appeared first on the face and trunk. Moreover, there were usually more lesions on the face and trunk than on the extremities. The term centrifugal could in no way be applied to the rash in any of these patients. Whereas our first patient afforded a good illustration of this rash distribution, the eruption in the second was clearly of a centrifugal character. The hemorrhagic forms of the disease may initially be mistaken for measles *(4)*. A fulminating meningitis can also be simulated and in a series of 13 such cases *(5)* this proved to be the most frequent initial diagnosis. One of these 13 patients was believed to have a "surgical abdomen" and an appendectomy was performed. In case 2 lower abdominal pain

(4) Illingworth, R. S., and Oliver, W. A.: Smallpox in Middle East; lessons from 100 cases. Lancet 2: 681-685, Nov. 25, 1944.

(5) Boeck, V. H. F.: Smallpox among U. S. soldiers in Korea. Bull. U. S. Army M. Dept. 6: 45-58, July 1946.

was a major symptom, but because of the other severe widespread symptoms and the absence of abdominal findings, attention was not focused on the abdomen.

The leukocyte count, which was 10,700 initially in case 1, fell to 6,950 following hydration. Both patients had a mild leukocytosis during the pustular stage, a gradual increase in the mononuclear elements as the disease progressed, and an eosinophilia during the period of crusting.

In the treatment of smallpox, fluid replacement to offset the severe dehydration is of greatest importance. It is doubtful whether the first patient, whose urine specific gravity was 1.035, on admission, and who continued to vomit for 36 hours thereafter, would have survived without it. Although neither patient required blood transfusion, such support is usually necessary in patients with the frequently fatal hemorrhagic type of the disease. In the presence of toxic myocarditis, the patient should be digitalized. If the skin permits, mild antiseptic and antipruritic solutions may be used locally.

Neither aureomycin nor chloramphenicol in combination with penicillin had any discernible effect on the expected course of the disease in these patients, except insofar as they undoubtedly prevented secondary complications and made possible the uneventful course which was seen in each patient after the height of the disease was reached. In case 1 a total of 11 grams of aureomycin and 2,400,000 units of penicillin did not prevent the height of the pustular stage with its elevation in temperature and leukocyte count from being attained. Similarly, in case 2 the pustular stage, with a concomitant secondary fever and leukocytosis, was manifested, despite the giving of a total of 10 grams of chloramphenicol and 4,200,000 units of penicillin. These doses represent only the amounts of antibiotics given in the hospital as of the day of the secondary fever peak because medication given beyond this point, although important in controlling and preventing complications, could have little or no effect on the primary virus infection which normally is quiescent after the secondary fever is passed and crusting begins. Penicillin has been used successfully in the treatment of smallpox to prevent secondary infection, but, as might be expected, with no effect on the primary disturbance *(6-8)*. The amounts of aureomycin and chloramphenicol that we used were admittedly small. It remains to be seen whether earlier administration or larger doses of these antiviral agents will have any effect on the virus of variola.

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(6) Jeans, W. D.; Jeffrey, J. S.; and Gunders, K.: Penicillin and smallpox; report of 4 cases. Lancet 2: 44-45, July 8, 1944.

(7) Weeks, K. D., and McClelland, W. S.: Hemorrhagic smallpox; report of case with recovery; treatment with massive doses of penicillin. U. S. Nav. M. Bull. 47: 707-715, July-Aug. 1947.

(8) Agerty, H. A.: Outbreak of smallpox in American military personnel in Japan. New England J. Med. 237: 305-309, Aug. 28, 1947.

With the movement of more and more Americans into regions of endemic and epidemic smallpox, it becomes increasingly important for medical personnel to learn to recognize and treat this disease. Of greater importance is its prevention. Increased emphasis should be placed on proper vaccination. Unfortunately, as is evident from this report, the procedure is not always given the attention it demands. A successful vaccination program must include: (1) the use of a potent vaccine which has been properly stored at temperatures below freezing; (2) correct performance of the vaccination; and (3) careful interpretation of the reaction. The occurrence of smallpox in supposedly immunized persons undoubtedly stems from the use of an impotent lymph or a faulty technic of administration. The contention that such cases are due to the inability of a certain vaccine to protect against an especially virulent strain of the virus is remote *(9)*. In support of this may be cited the outbreak of smallpox in Japan and Korea during 1945-46, which was rapidly brought under control among the military personnel by means of revaccination with a potent preparation even though cases continued to occur in the civilian population *(10)*. It should be remembered that a potent vaccine will produce vaccinia in all unvaccinated persons and an accelerated reaction in 50 percent of those who have not been vaccinated or had smallpox in the previous 10 years *(11)*. Either of these reactions is proof of a successful immunization. The immune reaction, better called the immediate reaction because it can be mimicked by a number of other things, including trauma and sensitivity to lymph protein, can be considered to represent true immunity when the vaccine used is known to have produced a primary or accelerated reaction. The vaccination site should be checked at least twice so that accelerated reactions, which may initially appear to be immediate reactions, will not be misinterpreted. When no reaction is found, the only permissible conclusion is that there has been no reaction and the person should be revaccinated.

SUMMARY

Although aureomycin and chloramphenicol in conjunction with penicillin failed to influence the natural course of smallpox in 2 patients who had been vaccinated shortly prior to the onset of the disease, they were of great value in preventing secondary complications. Careful vaccination is essential if smallpox is to be completely eliminated among military personnel.

(9) Horgan, E. S., and Haseeb, M. A.: Vaccinia virus; immunological unity of different strains. Lancet 2: 170-171, Aug. 11, 1945.

(10) Smadel, J. E.: Smallpox and vaccinia. In Rivers, T. M. (editor): Viral and Rickettsial Infections of Man. J. B. Lippincott Co., Philadelphia, Pa., 1948. Chap. 15, pp. 314-336.

(11) Leake, J. P.: Questions and answers on smallpox and vaccination. Pub. Health Rep. 42: 221-238, Jan. 28, 1927; also U. S. Nav. M. Bull. 25: 461-475, Apr. 1927.

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EXHIBITS ACCEPTED FOR PRESENTATION AT CHICAGO SESSION OF AMERICAN MEDICAL ASSOCIATION 9 to 13 June 1952

Experimental Medicine and Therapeutics Section:

Changing Patterns in the Susceptibility of Microorganisms to Antibiotics—Col. Dwight M. Kuhns, MC, U. S. A.

Military Medicine Section:

Study on Enteric Disease by the Joint Dysentery Unit—Col. William S. Stone, MC, U. S. A.

U. S. Air Force Air Crew Effectiveness Program—Maj. J. A. Connors, U. S. A. F. (MC)

In Flight Feeding and Nutritional Problems in Military Medicine—Col. Robert H. Blount, U. S. A. F. (MC)

Arteriovenous Fistulae and False Aneurysms in Korean War Wounded—Brig. Gen. Sam F. Seeley, MC, U. S. A.

Polyethylene Cranioplasty—Capt. Walter S. Lockhart, Jr., MC, U. S. A.

The Army Medical Library—Lt. Col. F. B. Rogers, MC, U. S. A.

The Exposure Treatment of Burns—Col. William H. Amspacher, MC, U. S. A.

Virus Diseases of Special Interest to the Dermatologist—Capt. Ernest R. Trice, MC, U. S. A.

Ideal Amputations and Prostheses—Col. A. W. Spittler, MC, U. S. A.

Normal and Abnormal Human Heredity—Dr. Henry W. Edmonds, Armed Forces Institute of Pathology

Preventive Medicine and Industrial Medicine and Public Health Section:

Malaria—Dr. Ernest Carroll Faust

U. S. Air Force Preventive Medicine Program—Col. Fratis L. Duff, U. S. A. F. (MC)

Radiology Section:

Malignant Primary Tumors of Bone—Dr. Lent Johnson

Total Body Ionizing Radiation—Brig. Gen. Elbert DeCoursey, MC, U. S. A.

Scientific Section:

The Use of Small Laboratory Animals in Radiobiology—Friedrich T. Ellinger, M. D., Naval Medical Research Institute, National Naval Medical Center, Bethesda, Md.

Urology Section:

Subcapsular Nephrectomy—Col. J. C. Kimbrough, MC, U. S. A., and Capt. W. H. Morse, MC, U. S. A.

The Veterinarian in Civil Defense[1]

Frank A. Todd, *D. V. M.* [2]

THE purpose of this article is to present one of the major responsibilities of the veterinary profession in civil defense, to describe the organization available to conduct this responsibility, and to indicate how the civil defense director and the civil defense health services officer should use the services of the veterinarian for a more complete protection of human and animal health in an emergency. We depend on farm animals and the farm crops for our food, our clothing, and certain biologic products. The veterinarian is responsible for the protection of domestic animals against disease as well as for the protection of human beings against those hazards which result from contact with diseased animals and the consumption of tissues or products of diseased animals. Most of the disease agents listed as potential weapons of biologic warfare infect both man and animals. These agents are significant in that they are normally transmissible from animals to man and constitute one of the major unsolved problems of communicable disease control in public health today.

The veterinarian on State and local civil defense staffs, as a consultant to the civil defense director and the civil defense health services officer, coordinates the civil defense efforts of all veterinary organizations and related agencies interested in animal disease control. The civil defense health services officer should use the veterinarian's knowledge of the animal-man disease relationship, his experience in disease control, and his ability to extend the epidemiologic investigations to include the animal and animal food resources of human disease. The veterinary practitioners, as well as those engaged in the inspection of animal food products, can provide animal disease information that might be the first indication of biologic warfare against man or animals. In addition, the veterinarian can and should be used

(1) Presented at the Medical Civil Defense Conference, Chicago, Ill., November 9-10, 1951, sponsored by American Medical Association, American Hospital Association, and the Association of State and Territorial Health Officers.

(2) Consultant, Veterinary Services, Health and Special Weapons Defense Division, Federal Civil Defense Administration, Washington, D. C.

in the preparation and teaching of civil defense educational and training material for all members of the health and special weapons defense services and for the general public.

The United States Public Health Service has recently initiated a sectional research program in microbiology dealing with improved diagnostic technics and disease control measures. This program includes work on diseases transmissible from animals to man. The service of the laboratories investigating human disease is augmented by veterinary laboratories of State experimental stations, veterinary colleges, or State diagnostic laboratories, known as collaborating laboratories. This program is to encourage more definite relationship between investigators and official health agencies which are responsible for the control of communicable diseases.

The Bureau of Animal Industry of the United States Department of Agriculture and the United States Public Health Service have been charged with communicable disease control; the former for animal diseases and the latter for the diseases of man. The Bureau of Animal Industry operates on the national level, dealing with national or interstate problems, and co-operates within the States in animal disease control programs. The State livestock sanitary officials, like the health departments, are concerned with disease problems within their respective States. This combination of Federal and State organizations for animal disease control functions in practically every livestock-raising community of the country.

The local veterinary practitioner with his experience and knowledge of the usual diseases and the normal incidence of disease in his community will probably be the first person to realize that there is an unusual increase in the incidence of any disease or that a new type of disease is present. When unusual diseases or conditions are detected by the veterinary practitioner, he immediately reports his suspicions to the State veterinarian. In the case of vesicular diseases, such as foot-and-mouth disease or vesicular stomatitis, specially trained diagnosticians of the Bureau of Animal Industry will be called to conduct the necessary investigations, to establish the diagnosis, and to instigate the required control measures. Epizootiologic investigations are initiated at once to determine the source of infection and the possible extent of the outbreak. The chief of the Bureau of Animal Industry, in Washington, is notified by the local Bureau official at the time the outbreak is initially reported. The chief is kept informed of all operations and their results. For those diseases other than the vesicular type, aid for establishing a diagnosis will be sought from the collaborating laboratories.

Foreign animal diseases have gained entrance into this country in the past despite peacetime inspection and quarantine measures. They have, in each case, been eradicated by strict quarantine and destruction and disposal of infected and exposed animals, followed by thorough

disinfection. It must be recognized, however, that if foreign diseases were to be introduced into this country by an enemy, the success of such action would depend on these diseases being widely scattered. If such outbreaks were numerous and simultaneous, modifications of prior control measures might become necessary. Such modified procedures might of necessity include the use of vaccines or other biologic products. Stockpiles of biologic products for some of these diseases are available. In the case of foot-and-mouth disease, there are several types of virus that cause the disease and within these types are several variants. The types and variants are difficult to identify and a vaccine effective against one type is ineffective against another. Moreover, the types and variants can be identified only by personnel with a high degree of technical training, extensive experience, and adequate facilities. This country has the trained personnel but lacks the facilities for identifying the causative virus and for vaccine production. Federal laws prohibit research on this disease within the continental limits of the United States. The recent co-operative control program between the United States and Mexico, for the eradication of foot-and-mouth disease from the latter country, has provided extensive experience for American veterinarians in controlling this infection.

The animal diseases that are usually discussed as potential biologic warfare agents for use against the domestic animals of this country include rinderpest, foot-and-mouth disease, Teschen disease, fowl plague, and Newcastle disease.

Rinderpest is a viral infection of ruminants, characterized by an acute croupous diphtheritic inflammation of all mucous membranes. Because of a mortality of 75 to 90 percent it is one of the most serious and devastating diseases of cattle.

Foot-and-mouth disease is a highly communicable viral disease affecting all cloven-hoofed animals and is characterized by the formation of vesicles on the tongue, buccal mucosa, the udder, the coronary cushion, and the interdigital spaces of the feet. Although the mortality is relatively low, the disease causes a significant decrease in meat and milk production.

Teschen disease has become a serious problem in the swine industry of several of the Eastern European countries. It resembles, but is immunologically distinct from, poliomyelitis.

Fowl plague and Newcastle disease cause extremely high mortality in most of the barnyard fowls. Newcastle disease is transmissible to man, attacking the tissues of the lungs, nerves, and endothelium.

There are other foreign diseases of livestock that could cause great losses of the domestic animals of this country if successfully introduced. Experimental data and information available reveal that some are difficult to transmit experimentally, but, because man is capable of altering the characteristics of a micro-organism by selecting variants

with increased resistance and pathogenicity, we should not overlook the possibility of their introduction into this country.

Our programs for controlling the diseases already present in our domestic animals must be continued and improved. Our present research on these diseases is directed toward more effective vaccines with longer-lasting immunity. Some of these diseases, such as hog cholera, anthrax, brucellosis, swine erysipelas, and Newcastle disease, could cause as much damage as a foreign disease if allowed to run unchecked through our farm animals. Each of these diseases, except hog cholera, is transmissible from animal to man.

A recently completed booklet, entitled "What the Farmer Should Know About Biological Warfare," points out the farmer's responsibilities and suggests that he immediately report unusual increases in animal diseases or the appearance of unusual animal diseases to his veterinarian. This personal responsibility is consistent with principles included in a technical manual we are now developing on defense against biologic warfare. This manual is for the use of physicians, veterinarians, and other technical personnel.

The success of our civil defense efforts in preventing and controlling animal diseases depends on strengthening and expanding our present facilities and programs. Such additional activities will include:

1. Assuring that those agencies responsible for disease control have adequate facilities for studying and conducting research on those diseases exotic to the United States.

2. Improving and expediting present methods for accurate diagnosis.

3. Establishing an effective country-wide system of reporting outbreaks of animal disease. This system must include evidence of increased incidence of any disease as well as the appearance of any unusual disease.

Lipid Pneumonia[1]

I. Louis Hoffman, *Colonel, U. S. A. F. (MC)*

Horace S. Bell, *Captain, U. S. A. F. (MC)*

LIPID pneumonia is a form of pulmonary consolidation, usually chronic, which may follow the entrance of fats or oils into the lungs (2). Waters et al. (3) found that iodized olive oil placed in the lungs of dogs was followed by a proliferative bronchopneumonia which was often fatal. Brown (4) and others pointed out that bland oils remained in the lung for a long time and were slowly absorbed by large mononuclear cells. Olive oil and liquid petrolatum introduced into the lungs cause a chronic proliferative pneumonia and other oils, such as chaulmoogra oil, produce a rapid and violent pulmonary inflammation. Lipid pneumonia was first described in children by Laughlen (5) and Pinkerton (6,7) soon added more cases to the literature (2).

Etiology. Introduction into the alveoli of any lipoid material, whether of mineral, animal, or vegetable origin, may cause pneumonia. Some of the lipoid substances reported as causing lipid pneumonia were mineral oil, chaulmoogra oil, cod liver oil, milk fat, vaseline, lard oil, egg yolk, olive oil, sesame oil, poppy-seed oil, and, rarely, iodized vegetable oil used in pneumoroentgenography. Secondary infection may be caused by material from the upper respiratory tract carried downward into the alveoli by the lipoid material. Infancy; debilitation; old age; diseases of the central nervous system effecting deglutition; feeding or swallowing difficulties; bland, oily nose drops that may gravitate from

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(1) Reese Air Force Base, Lubbock, Tex.

(2) Goodwin, T. C.: Lipid pneumonia. In McQuarrie, I. (editor): Brennemann's Practice of Pediatrics, vol. II. W. F. Prior Co., Inc., Hagerstown, Md., 1948. chap. 49, pp. 1-5.

(3) Waters, C. A.; Bayne-Jones, S.; and Rowntree, L. G.: Roentgenography of lungs. Arch. Int. Med. 19: 538, Apr. 1917. Cited in footnote reference (2).

(4) Brown, A. L.: Fate of iodized oil (lipiodol) in lungs. Surg., Gynec. & Obst. 46: 597-601, May 1928. Cited in footnote reference (2).

(5) Laughlen, G. F.: Pneumonia following naso-pharyngeal injections of oil. Am. J. Path. 1: 407-414, July 1925. Cited in footnote reference (2).

(6) Pinkerton, H.: Oils and fats; their entrance into and fate in lungs of infants and children; clinical and pathologic report. Am. J. Dis. Child. 33: 259-285, Feb. 1927. Cited in footnote reference (2).

(7) Pinkerton, H.: Reaction to oils and fats in lung. Arch. Path. 5: 380-401, Mar. 1928. Cited in footnote reference (2).

the nasal pharynx to the larynx and thence to the lungs without eliciting a cough reflex; mechanical defects of the palate; tracheotomy; vomiting; and gavage may predispose to this condition.

Pathogenesis and pathology (2,8-10). Lipid pneumonia is predominantly localized in the central and lower portions of the lungs, especially in the basal circumhilar and paramediastinal segments of the lower lobes. Occasionally the base of the middle lobe of the right lung is implicated. Involvement of the upper lobes is not common. The posterior portions of the lower lobes are more markedly affected than the anterior portions. The disease is usually bilateral and frequently it is roughly symmetrical, but the earliest and most extensive involvement is likely to be on the right.

Vegetable oils produce a mild chronic inflammation with phagocytosis and usually absorption of the fat. Animal oils are first emulsified in situ and later attacked by phagocytes. Some of these reach the lymph nodes where fibrosis results after 2 or 3 months and others remain in the lumina of alveoli where fibrosis also results. Foreign body giant cells are found wherever the fat is. Fatty acids produced by hydrolysis are the irritative agents and the extent of the damage depends on the proportion of free fatty acid in the original oil and on the rate of its hydrolysis. Chaulmoogra oil produces acute tissue necrosis from the beginning. Mineral oils are not hydrolyzed but produce an immediate phagocytic response in the alveolus. They are rapidly emulsified and some of the droplets are transferred to the lymph nodes. Fibrous tissue reaction occurs both in the alveoli and lymph nodes, but more slowly than with animal oils. Mineral oil in the lung usually results in proliferative pneumonia. The pleura is usually smooth. Extensive, firm consolidation is felt beneath its surface. The lungs feel heavy. Nodules can be palpated throughout the involved area and lobules, solid with pneumonia, are adjacent to or almost surrounded by normal, air-containing tissue, or emphysematous lung. The greater part of the consolidation is nearer the hilum and in the posterior part of the lung. The gross lesions vary from a generalized consolidation resembling red hepatization to local areas of fibrosis surrounding a mass of necrotic tissue and oil which ranges in size from that of a milium to that of a paraffinoma several centimeters across. Several observers have recorded fibrotic nodules in liver, lungs, spleen, kidneys, adrenals, and ovaries. Some observers found fibrosis extensive enough to produce arteritis and periarteritis in the kidney with extensive glomerular damage. The septums of the lung are thickened. Many of the nodular areas when cut look like tubercles. The cut surface of the

(8) Rewell, R. E.: Lipoid pneumonia; pitfall in diagnosis. Brit. M. J.1: 409-411, Mar. 29, 1947.
(9) Kirklin, B. R.: Lipoid pneumonitis. Radiology 35: 261-267, Sept. 1940.
(10) Proper, S. A.: Lipoid pneumonia. Brit. M. J. 2: 282-283, Aug. 31, 1940.

lung feels greasy and when viewed under ultraviolet light may show a fluorescence characteristic of the oil involved.

Microscopically an acute or subacute bronchitis is almost always present. Globules of oil are free in the bronchi and fat-laden mononuclear phagocytes may be seen traveling to the draining lymph nodes. The wall of the alveolus is thickened and the avelolar space is packed with large mononuclear cells containing small droplets of fat. There may be an inflammatory exudate of red blood cells, fibrin, and polymorphonuclear neutrophils in some of the alveolar spaces. The tubercle-like nodules are the result of tissue reactions around large masses of fat, particularly such fat as may cause necrosis. The center of the mass is emulsified fat in the midst of caseous material. Giant cells and epithelial cells are present. Calcification may occur *(2)*.

Lipid pneumonitis plus bronchopneumonia is a grave combination and is often fatal, especially in infants. On the other hand, in a mild to severe case of uncomplicated lipid pneumonitis, restoration to normal may follow quickly when further aspiration of lipoid material is stopped and postural drainage is instituted. In chronic cases, anatomic restitution cannot be expected because of the extensive fibrosis and contraction.

The clinical findings (2) are often colored by an accompanying condition. The onset of lipid pneumonia is insidious unless a large aspiration occurs. Infants with extensive consolidation do not respond well to treatment. Their development is also retarded. Rapid respiration is characteristic but dyspnea is uncommon. There may be a dry nonproductive cough. Cyanosis is rare; clubbing of the fingers has been observed. Fever does not occur in children with uncomplicated lipid pneumonia. The physical signs are fewer than one would expect from the extent of the lung involvement. Dullness to percussion over the affected area and suppressed bronchial breathing are found. Moist rales may occur. The physical signs are more often posterior and on the right side. Repeated roentgenograms are the greatest aid in diagnosis because they show the extent and chronicity of the lesion. The density of the x-ray shadow is likely to be greatest near the hilum on the right side, becoming less marked, irregular, and spotty in character toward the periphery.

The complications and residua are (1) delayed growth; (2) emphysema; (3) lung abscess; (4) bronchopneumonia; (5) empyema; (6) purulent bronchiolitis; (7) bronchiectasis; and (8) increased susceptibility to secondary bacterial invasion due to repeated aspiration of oil and other infective material, the serious interference with the normal self-cleansing action of the lung brought about by choking of the macrophages with oil, the blockage of the lymphatics, the disturbed ciliary action, the increasing fibrosis and, in some cases, the underlying chronic passive congestion.

Diagnosis (2,9,11,12). Unless one knows the cause and circumstances of the case, the clinical diagnosis is not based on any distinctive features but on a combination of facts. In suspected cases the sputum should always be examined for free oil droplets or large phagocytic monocytes containing fat or oil. Aspiration biopsies for histologic examination and specific fat stains for identification may be performed. In the differential diagnosis, tuberculosis, pulmonary fungus or spirochetal disease, primary atypical pneumonia, and chronic pulmonary infections such as bronchiectasis, lung abscess, and bronchopneumonia should be ruled out.

Prevention and treatment (2,11). There is no specific treatment. The best therapy is prevention. Every physician should impress on his patients that care and common sense should be practiced in the use and in the method of administration of oil and lipoid materials. No harm is done provided the patient is in good general condition and provided self-administration is carried out correctly or under medical supervision. The agent preferably should not be administered before retiring because the material may trickle down into the trachea and bronchi and insidious aspiration may occur in this fashion during sleep. If the patient is confined to bed, flat on his back, utmost caution should be exercised in the amount, frequency, and duration of administration. In patients with subdued or absent reflexes from whatever cause, other substances should be substituted for lipoid agents. Lipoid agents should be discontinued in the presence of symptoms favoring aspiration. This is particularly true in severe debilitating diseases and those conditions affecting the normal reflexes of the throat and of deglutition.

In patients suspected of having pneumonolipoidosis, the use of iodized oil for bronchography should be deferred. In infants with conditions favoring aspiration, it is best to use fat-free formulas. The main therapy consists of adequately treating the associated disease or disorder. Bronchoscopy as a therapeutic measure is of little value. Radiotherapy should be avoided because it may augment existing fibrosis. Activity should be reduced to conform to the disability resulting from the pulmonary involvement. Fatty liquids should be kept out of the reach of children. The condition rarely occurs in strong, healthy children. In premature, weak, or debilitated infants, and in older children who for any reason have a diminished cough reflex, precautions must be taken to avoid the aspiration of fatty material. Oily nose drops and sprays should be avoided; mineral oil should not be given by mouth; and when cod liver oil is poorly taken, it should be replaced by a vita-

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(11) Bishop, P. G. C.: Oil aspiration pneumonia and pneumolipoidosis. Ann. Int. Med. 13: 1327-1359, Feb. 1940.

(12) Nathanson, L.; Frenkel, D.; and Jacobi, M.: Diagnosis of lipoid pneumonia by aspiration biopsy. Arch. Int. Med. 72: 627-634, Nov. 1943.

min concentrate. Gavage and difficult feeding should be done only by experienced persons. When oil is given to weak infants, they should not be left in a supine position.

CASE REPORT

A 1-year-old girl was admitted to the hospital on 11 May 1951 with a history of having swallowed 120 cc. of furniture polish. In the act of swallowing she also coughed and vomited. On reaching the dispensary her stomach was washed out with normal saline solution and the washings contained some orange-colored oily material similar to the furniture polish swallowed. On admission the child was acutely ill, pale, and dyspneic. Because her inspirations and expirations were labored she was placed in an oxygen tent with the foot of the bed raised in order to encourage postural drainage. Roentgenograms of her chest on admission were normal. Her course in the hospital was characterized by an acute febrile episode accompanied by marked dyspnea and prostration. Her temperature fluctuated between 100° and 105° F. for 11 days and then gradually subsided, becoming normal on the nineteenth day.

The dyspnea was marked during the first 2 weeks and rapidly subsided from then on. The pneumonic process was discovered by physical examination on the second hospital day and was characterized by suppression of breath sounds over the lower portion of both lungs and moist crepitant rales over the lower lobe of the right lung, extending a day later to the lower lobe of the left lung. There was also an increase in both vocal and tactile fremitus over the lower lobes of both lungs. Roentgenograms taken on the third hospital day showed a homogeneous consolidation of the lower lobe of the right lung and a dense, patchy infiltration of the lower lobe of the left lung extending to the hilus. The child remained intermittently in the oxygen tent for 2 weeks and was given 600,000 units of aqueous procaine penicillin intramuscularly every day. Because aureomycin and chloramphenicol given orally were not tolerated, 1 gram of chloramphenicol was given intramuscularly every day. The antibiotic therapy was discontinued on the fifteenth hospital day. Supportive therapy, including adequate diet, multivitamins, and iron, was also instituted.

Roentgenograms of the chest taken on 18 May showed an increase in irregular infiltration of the lower lobes of both lungs. From the fifteenth hospital day on the patient recovered rapidly and was discharged on the twenty-first hospital day. Her lungs gradually cleared, according to physical and roentgenographic findings, and by 16 July they were normal. It was determined from the manufacturer that the furniture polish contained a mineral-seal oil base with coloring and aromatics added.

DISCUSSION

This case is presented primarily as an acute case of lipid pneumonia with recovery. Most patients reported in the literature had been exposed

to a lipoid agent over a prolonged period of time. The usual process of exposure or contact was the ingestion of mineral oil or oily nose drops. In our patient it was apparent that there was a sudden, almost overwhelming exposure due to aspiration of an oil-containing furniture polish which consists of a mineral-seal oil base with aromatics and coloring. Aspiration in this case evidently occurred during vomiting of the polish, as is not uncommon in small children. The diagnosis did not present a problem by virtue of the dyspnea, physical findings, roentgenograms, and the knowledge of the etiologic agent. Treatment consisted of oxygen for the dyspnea and hypoxia, postural drainage, antibiotics to prevent or treat any pulmonary infection, and supportive therapy. It is believed that the pulmonary lesion in this patient was due to the irritant effect of the aspirated polish plus pathogenic organisms which were either aspirated with the oil or present as secondary invaders. The child was so ill that recovery was doubtful until a week had passed. It was surprising that the pulmonary lesion cleared up without any residua.

BOOK REVIEW

Urine and the Urinary Sediment, A Practical Manual and Atlas, by *Richard W. Lippman,* B. S., M. D., Research Associate, Institute for Medical Research, Cedars of Lebanon Hospital, Los Angeles, Calif.; Fellow of the John Simon Guggenheim Memorial Foundation. 124 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $7.50.

This monograph consists of 3 chapters dealing with proteinuria, elements of the urinary sediment, observations on the urine in disease, general considerations, and technic. Many of the illustrations consist of color photographs of material in an ordinary hemocytometer chamber taken with the high-dry objective. These preserve subtle differences in shade and refraction that make them recognizably similar to actual microscopic appearances. As the result of the author's association with Dr. Thomas Addis, many of the interpretive comments concur with Dr. Addis's opinions and observations. The monograph serves as a practical guide in the clinician's examination of urine and urinary sediment and as a record of methods and interpretations that have evolved during a long period of careful observation. This book would be a particularly valuable addition to the library of the clinical pathologist, the internist, and the urologist.—*Col. L. K. Mantell, MC, U. S. A.*

Surgical Correction of Ankyloglossia

James G. Rogers, *Lieutenant Commander, DC, U. S. N.*

Bruce L. Douglas, *Lieutenant, junior grade, DC, U. S. N. R.*

ANATOMIC, practical, and surgical considerations are important in the treatment of ankyloglossia.

Anatomic and embryologic considerations. The inferior surface of the tongue is covered by smooth, thin mucosa which is reflected to the lingual gingivas and the floor of the mouth. When the tongue is raised, an elevated fold of this mucosa, the lingual frenulum, can be seen in the midline. A tongue-tied condition (ankyloglossia) is caused by a short frenulum attached far forward to the floor of the mouth or the lingual gingiva. This limits the movement of the tongue to varying extents. On either side of the frenulum, a ridge of fimbriated mucosa extends forward and medially toward the tip of the tongue. Between these ridges and embedded in the ventral surface of the tongue are the lingual nerves and vessels (1). The root of the tongue is derived largely from hyoid arch material, on which the rudiment known as the copula appears. Additions to the root are made from the third and perhaps the fourth branchial arches. As this combination of separate components enlarges, the body portion gradually becomes free from the sides and the floor of the oral cavity. The lingual frenulum is thus formed. It varies greatly in individual patients in length and place of attachment, extending from the lingual gingiva or the floor of the mouth to the ventral surface of the tongue near or not far from the tip (2).

Practical considerations. The lingual frenulum is important in various physiologic functions of the tongue. Its length and place of attachment limit the movement of the tongue in speech and mastication. Speech is affected when there is limitation of extension or elevation of the tip of the tongue or when there is an inability to touch the palate

(1) Shapiro, H. H.: Applied Anatomy of Head and Neck for Students and Practitioners of Dentistry. 2d edition. J. B. Lippincott Co., Philadelphia, Pa., 1947.

(2) Arey, L. B.: Developmental Anatomy. 5th edition. W. B. Saunders Co., Philadelphia, Pa., 1946.

with the dorsum of the tongue. After a period of time, most patients with ankyloglossia, though often aware of their handicap, are able to compensate sufficiently to pronounce many sounds properly or nearly so. There are others who, for one reason or another, are unable to effect this compensation. Occasionally the high attachment of a lingual frenulum to the gingiva may interfere with the proper construction of a full lower denture.

Surgical considerations. In attempting to free the tongue from limitation of movement, the proximity to the floor of the mouth and the possibility of introducing infection into the fascial planes of the neck must be considered. Therefore, it is advisable to free the frenulum at one or both ends rather than take what may seem to be the easier approach—to clip or sever the membrane in the middle. The operation

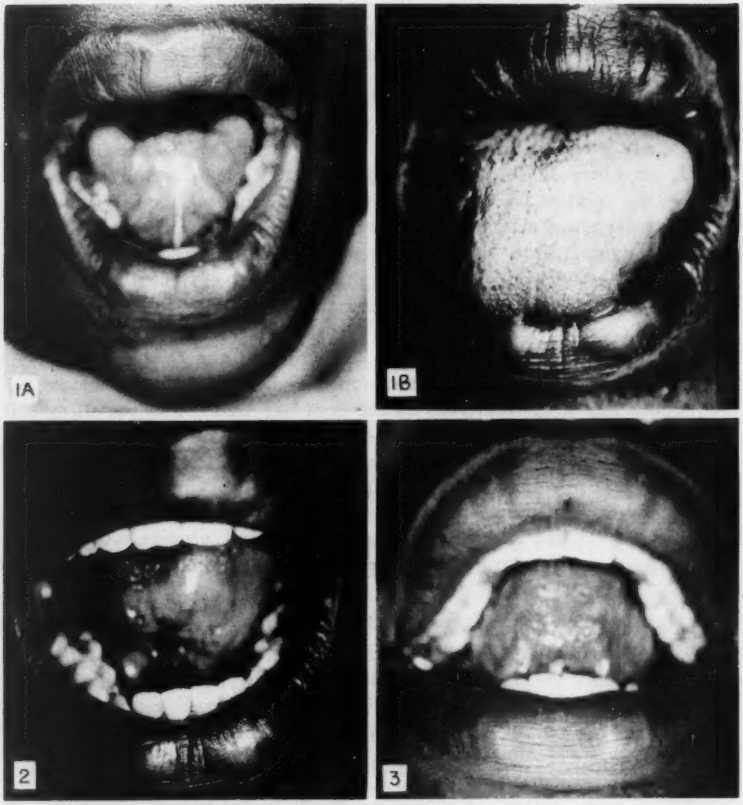

Figure 1. Photographs taken before operation showing limitation of (A) *upward extension of tongue and* (B) *forward protrusion of tongue. Figure 2. Photograph taken immediately after operation. Figure 3. Photograph taken 1 week after operation.*

is neater and gives a better result when the latter technic is avoided. Approximating anatomic structures should be visualized before attempting any surgical procedure in this area.

CASE REPORT

In a routine examination at the dental dispensary of the Marine Corps Recruit Training Depot, Parris Island, S. C., a 19-year-old Negro with a marked obstruction of lingual function was seen. An extremely short lingual frenulum was attached high on the gingiva between the lower central incisor teeth and close to the apex of the tongue on its under surface (fig. 1). The patient reported that he had always had difficulty in moving his tongue and there was a noticeable impairment of speech. He could not pronounce the guttural "k" or "g" because the proper enunciation of these sounds requires the back of the tongue touching the soft palate. Though he was capable of placing his tongue in the proper positions for the formation of "s" and "x," the sound was incorrect. His "l's" were materially affected because he could not extend his tongue at all and they ended with more of a "w" sound.

Under local infiltration anesthesia, lingually into the floor of the mouth, hugging the bone, and into the ventral tip of the tongue, a narrow, but long, V-shaped incision was made, freeing the gingival attachment of the frenulum. A similar incision was made, as the tip of the tongue was firmly grasped, along the lingual attachment. It was necessary to extend this incision about 1 inch toward the base of the tongue in order to insure adequate freedom of movement. The lingual incision was coapted and closed with 000 nylon sutures on an atraumatic needle (fig. 2). The tautness of the gingival tissue made closure of this incision impractical. The wounds were painted with tincture of benzoin. The sutures were removed 2 days after operation and the patient was instructed to exercise his tongue freely, particularly in extension and preferably in front of a mirror. One week following the operation, he was able to touch the lingual surfaces of his upper teeth and the rugae palatinae (fig. 3).

BOOK REVIEWS

Standard Nomenclature of Diseases and Operations, *Richard J. Plunkett,* M. D., Editor, and *Adaline C. Hayden,* R. R. L., Associate Editor. 4th edition. 1,034 pages. Published for the American Medical Association. The Blakiston Co., Philadelphia, Pa., publisher, 1952. Price $8.

This new edition is a comprehensive, well-organized work. Over 5,800 changes have been made to bring it up to date. The etiologic and topographic basis for diagnosis is used and diagnostic code numbers are given. Indentation of the book for quick reference assists the user. One

of the changes which increases the usefulness of the book and brings about a closer integration of the material presented correlates "Standard" diagnoses and code numbers with diagnoses and code numbers of the International Statistical Classification of Diseases, Injuries, and Causes of Death. The International List numbers have been included parenthetically in the body of the book and an appendix is supplied by which code numbers of the "Standard" and the "International List" may be properly cross-indexed by a hospital registrar.

The combined efforts of the best organized groups or committees of specialists on this continent and the standard nomenclature committee for that particular specialty has resulted in the present logical classification of nomenclature of diseases and operations. The revision of the section on diseases of the hemic and lymphatic systems made through close collaboration between the standard nomenclature committee and the committee for classification of the nomenclature of cells and diseases of the blood and blood-forming organs embodies the latest concepts and thinking on the subject. Major changes include a standardization of the names of the cells of the blood-forming tissues, and extensive revision and expansion of the classification of anemias, and leukemias, the latter being reclassified under "New Growths."

There have been a few changes to adapt the nomenclature of tumors to the standard coding system. The purpose of the new classification is to increase the ease and accuracy by which tumor data can be recorded, analyzed, and exchanged and thus to arrange for the calling of the same things by the same names. All commonly used diagnoses or commonly used synonymous terms for the tumors on the etiologic list have been included in order to facilitate the use of the tumor classification. The latter also provides additional information concerning tumors by supplying a malignancy code.—*Col. C. T. Young, MC, U. S. A.*

The 1951 Year Book of the Eye, Ear, Nose and Throat (October 1950-September 1951). The Eye, edited by *Derrick Vail*, M. D., D. Oph. (Oxon.), F. A. C. S., Professor and Director, Department of Ophthalmology, Northwestern University Medical School; Attending Ophthalmologist, Passavant Memorial Hospital and Cook County Hospital. The Ear, Nose and Throat, edited by *John R. Lindsay*, M. D., Professor of Otolaryngology, The University of Chicago, The School of Medicine. 456 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., publisher, 1952. Price $5.50.

This book reviews the literature in ophthalmology and otolaryngology covering the period from October 1950 to September 1951, inclusive. A short résumé of most of the important literature during that period is given. To the specialist it is an excellent review of the literature and to the general practitioner it is a quick reference to the modern trends. It is well indexed and edited. There are numerous instances where the editor has added his experience at the end of the reviewed article. Where indicated, excellent illustrations are reproduced.

—*Brig. Gen. E. Maxwell, U. S. A. F. (MC)*

Extensive Pericarditis Complicating Myocardial Infarction

James C. Syner, *Captain, MC, U. S. A.* (1)

THIS article reports a patient in whom acute hemopericardium developed coincidental to dicumarol therapy. The patient had sustained an extensive pericarditis complicating a posterolateral myocardial infarction. The experience is offered as a criterion for the contraindication of dicumarol, or the appreciation that such treatment is hazardous in this type of patient. To understand the natural history of a disease process and the events associated with the precipitation of an acute episode in its evolution, historical data must be accurate and detailed. This point proved to be of great importance in the diagnosis of the case to be reported. The history taken on admission was largely obtained from the immediate family and was misleading. It was only after repeated bedside interviews that the patient told what actually happened. Details, particularly of a psychogenic nature, are often passed off by the patient either as a function of personal reluctance or misunderstanding as to their fundamental importance.

CASE REPORT

A 64-year-old man, who had enjoyed good health until the onset of his present illness, had been retired from the Army in 1946 with findings of minimal generalized arteriosclerosis. His blood pressure at that time was 150/90 and, according to the historical data, he was asymptomatic. He had remained free of symptoms with no limitations on his activity during the period from retirement to the onset of his present illness. Throughout 6 December 1950, the day prior to the onset of his present illness, he felt well. He participated in varied activities, including a visit to his dentist who was treating a tissue irregularity on the gum margin. The patient was told that he should return the following day so that a biopsy of the tissue could be taken and studied for presence or absence of cancer. He stated that this

(1) Walter Reed Army Hospital, Washington, D. C.

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news had markedly lowered his spirits. He told his family nothing about this source of worry but brooded inwardly. Ignorant of his anxiety, his family saw his manifest introversion, disinterest in his favorite television program, and early retirement to bed that evening as the prodromal stage of an infectious disease. As they related the situation, infection was the chief consideration in the minds of the physicians treating the patient.

On 7 December he awoke complaining of midepigastric pain, pain over the anterior chest wall exaggerated on deep inspiration, and weakness. He passed 4 loose stools. His temperature was 101° F. Although he felt ill, he carried a coal hod weighing about 30 lb. from the cellar to an upstairs stove, experiencing marked weakness and profuse perspiration as a result. That afternoon he was seen by a physician who made a diagnosis of "intestinal flu," gave an injection of procaine penicillin and prescribed 500 mg. of aureomycin every 6 hours.

On 8 December the patient manifested increasing restlessness and complained of increased anterior chest pain associated with breathing. His temperature was 101° F. He complained of tenderness to touch along the right costal margin and severe pain radiating over the course of the sternocleidomastoid and trapezius muscles into the area of the mastoid and external auditory canal which he described as an "earache."

On 9 December his condition became worse. His temperature was 102° F. with no apparent response to the aureomycin and a total of 450,000 units of penicillin in the preceding 48 hours. On admission to this hospital his rectal temperature was 104° F.; pulse, 90; respirations, 28; and blood pressure, 137/76. He appeared to be acutely ill, complained of severe pain over the course of the superficial cervical muscles, aching joints, and inability to take normal breaths without sustaining severe anterior chest pain. Funduscopic examination revealed slight arteriosclerotic changes bilaterally but no hemorrhages, exudate, or papilledema. His chest was bilaterally symmetrical, presented no bulging or retraction of the intercostal spaces, but there was marked restriction of respiratory excursions. His lungs were clear to auscultation and percussion. On examination of his heart he complained of pain to light palpation over the precordium. On percussion the left border of cardiac dullness was 1 cm. outside the midclavicular line in the fifth interspace. The sounds were faint and muffled. No murmurs were heard. Over the left sternal border in the region of the third, fourth, fifth, and sixth interspaces a faint, high-pitched, scratchy, superficial friction rub, synchronous only with systole was heard. Examination of his abdomen revealed marked tenderness along the right costal margin, slight distention with a hyperresonant percussion note, and normal bowel sounds. His nail beds were cyanotic. He was placed in an oxygen tent and given symptomatic

therapy. A roentgenogram of his chest revealed generalized cardiac enlargement and clear lung fields. An ECG (fig. 1) showed changes compatible with an acute posterolateral myocardial infarction and associated acute pericarditis. Anticoagulant therapy was initiated and 300 mg. of dicumarol were given as soon as the base line prothrombin time was reported as 90 percent of prothrombin concentration (Quick method).

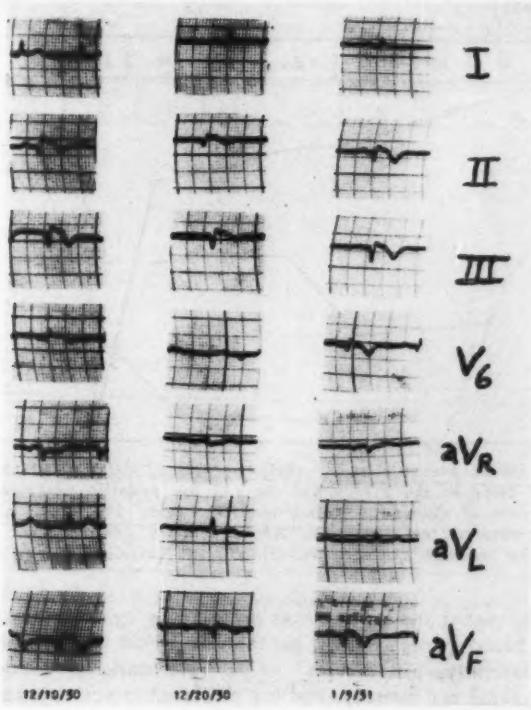


Figure 1.

On 10 December the precordial auscultatory findings changed in that a very loud, to-and-fro, superficial pericardial rub was now present. On 13 December a few coarse rales were heard at the base of both lungs and examination of the neck veins revealed moderate distention. A roentgenogram of the chest revealed a homogeneous density over the lower half of both lungs, suggesting a bilateral pleural effusion. Dicumarol was discontinued in view of the patient's marked reaction to relatively small amounts (fig. 2). On 14 December the friction rub had become extremely faint, cervical venous distention was much increased, the liver was palpated below the costal margin, and cyanosis was immediately evident on removing the patient from the oxygen

tent. The venous pressure without right upper quadrant pressure was 290 mm. (citrate) and with right upper quadrant pressure was 355 mm. (fig. 3). The arm to tongue circulation time was 45 seconds (decholin) and the arm to lung circulation time was 21 seconds (ether). On this day two doses of 0.4 mg. of lanatoside C were given. The subsequent presence of premature ventricular contractions prompted the discontinuance of this therapy.

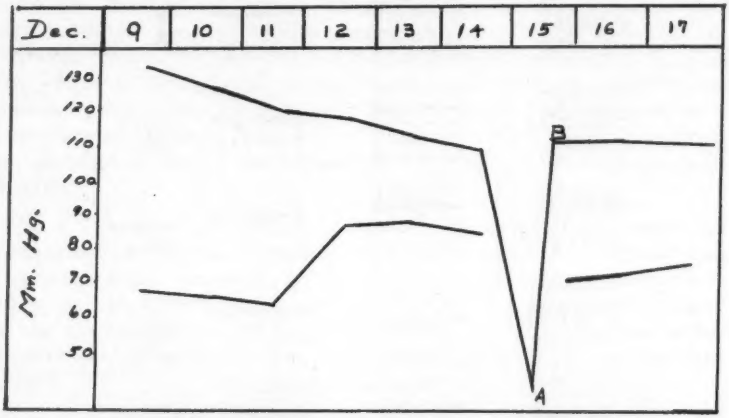


Figure 2. Blood pressure curve during period of critical illness showing that just prior to the pericardial tap (A) *the systolic pressure, obtained by palpation of the radial pulse, was less than 50. A diastolic reading was not obtained at this point. About 3 hours following the pericardial tap* (B) *the pressure had returned to the level existing prior to the critical episode.*

On 15 December the patient was noted to be cyanotic in the oxygen tent, his blood pressure was not obtained, his breath sounds were absent bilaterally, his cervical veins were markedly distended, his pulse was rapid and thready, and his prothrombin activity was reported as 9 percent concentration. A roentgenogram of the chest showed an increase in the homogeneous density in both lungs (fig. 4). A thoracentesis was performed and 150 cc. of dark brown fluid were withdrawn. The blood pressure by radial pulse palpation was less than 50. A diagnosis of cardiac tamponade was made and pericardial tap was decided on. Because of the risk of hemorrhage 72 mg. of hykinone were given intravenously and 1,000 cc. of blood were available for transfusion. A pericardial tap was performed and about 125 cc. of bloody fluid were withdrawn, a sample of which gave a hematocrit reading of 24. Following the pericardial tap a positive pressure oxygen mask set at 2 cm. with a flow of 9 liters of oxygen per minute was applied. Digitalization with lanatoside C was again started, and in the presence of premature ventricular systoles 200 mg. of quinidine

4 times daily were given. About 3 hours after the tap the patient manifested marked improvement. His color was excellent and he asked for food. A roentgenogram of his chest (fig. 5) revealed marked improvement in the lung fields with a 2-cm. diminution in the cardiac diameter. His pulse was full and regular with a rate of 62; his blood pressure was 115/70; and his heart sounds improved in quality. He passed more than 2,000 cc. of urine. From that time on he showed progressive improvement. Within 2 weeks the roentgenograms of his chest were entirely negative except for some generalized cardiac enlargement (fig. 6).

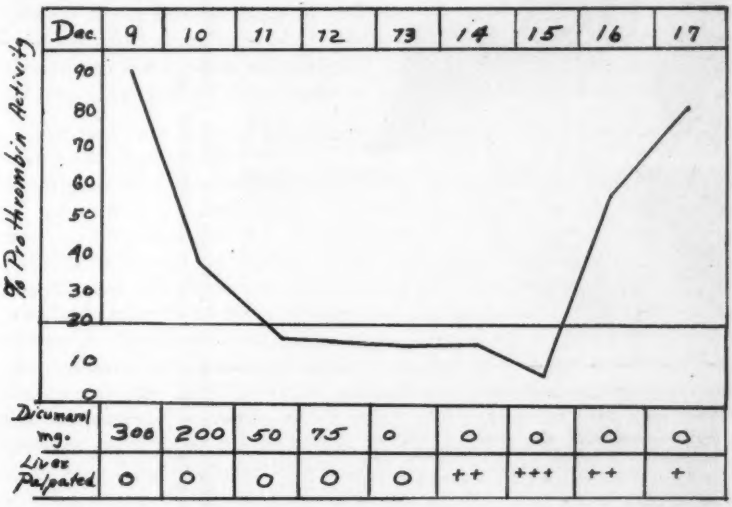


Figure 3. Relationship between prothrombin activity, dicumarol schedule, and liver enlargement. Just prior to the pericardial tap, the prothrombin activity was less than 9 percent.

DISCUSSION

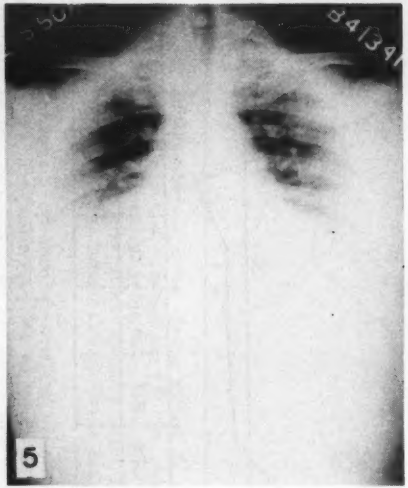
Myocardial infarcts frequently extend to the pericardial surface and occasionally an extensive fibrinous pericarditis results (2-4). The electrocardiographic differentiation may be especially difficult when cardiac infarction is associated with acute pericarditis as it is in at least 20 percent of patients (5). The combination of infarction with pericarditis is suggested by the presence of concordant S-T segment

(2) Cecil, R. L.: Textbook of Medicine. 7th edition. W. B. Saunders Co., Philadelphia, Pa., 1947.

(3) Lewis, T.: Diseases of the Heart Described for Practitioners and Students. 4th edition. Macmillan Co., New York, N. Y., 1946.

(4) Barnes, A. R., and Burchell, H. B.: Acute pericarditis simulating acute coronary occlusion; report of 14 cases. Am. Heart J. 23: 247-268, Feb. 1942.

(5) Friedberg, C. K.: Diseases of the Heart. W. B. Saunders Co., Philadelphia, Pa., 1949.



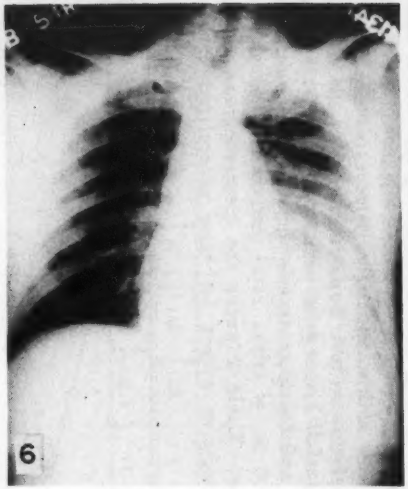


Figure 4. Roentgenogram taken prior to pericardial tap. Figure 5. Roentgenogram taken 2 hours following pericardial tap. Figure 6. Roentgenogram taken 3 weeks following pericardial tap.

elevation, as in isolated acute pericarditis, in association with Q waves in lead I and in the precordial leads or in leads II and III. Barnes and Burchell *(4)* observed that the characteristic early reciprocal deviation in RS-T segments in leads I and III seen in acute infarction may be overshadowed by such signs of pericarditis as elevation or dome-shaped upward rounding of RS-T segments in leads I, II, and III and that the only intact evidence of infarction in the early stages is the characteristic Q-wave pattern. Nichol *(6)* reported a review of serial ECG's taken on patients with pericarditis complicating myocardial infarction. He noted that when infarction of the posterior basal portion of the left ventricle is complicated by pericarditis, the electrocardiographic signs of this complication may be lacking, in contradistinction to the electrocardiographic pattern observed when pericarditis follows infarction of the anterior or apical portion of the left ventricle.

The fact that acute pericarditis may simulate acute coronary occlusion must also be considered *(4, 7, 8)*. Barnes and Burchell *(4)* reported 9 patients who presented this problem and emphasized the importance of serial ECG's in arriving at a correct diagnosis. In his series there were no traces of the T- or Q-wave pattern in the standard leads in the patients with uncomplicated acute pericarditis, whereas even though complicated infarction will reveal itself by T- or Q-wave patterns in the standard leads or by the appearance of a significant Q-wave or R-wave change in the apical precordial leads. Barnes and Burchell considered the tendency toward a rapid return to normal one of the most important features of the ECG in acute pericarditis, and distinguished it from that of coronary occlusion which tends to be reflected by recognizable changes for a much longer period. In the case presented the presence of an infectious pericarditis was strongly considered in the presence of a temperature which reached 104° F., historical data as originally obtained suggestive of an infectious disease, and the presence of an extremely loud pericardial friction rub. The problem which arose in this case relative to historical data has already been mentioned and subsequent clinical and laboratory data were not indicative of an infectious process.

It is a common belief that an intense friction rub is consistent only with the presence of an anterior wall infarction, but this is not the case *(5-9)*. Stewart and Turner *(9)*, reporting 60 patients with coronary thrombosis examined at necropsy, discovered that in 6 patients with generalized pericarditis who had an audible friction rub, the posterior surface was involved in 3.

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(6) Nichol, E. S.: Large pericardial effusion complicating acute coronary thrombosis. Ann. Int. Med. 11: 1900-1906, Apr. 1938.

(7) Wolff, L.: Acute pericarditis simulating myocardial infarction. New England J. Med. 230: 422-425, Apr. 6, 1944.

(8) Coffen, C. W., and Scarf, M.: Acute pericarditis simulating coronary artery occlusion. Am. Heart J. 32: 515-518, Oct. 1946.

(9) Stewart, C. F., and Turner, K. B.: Note on pericardial involvement in coronary thrombosis. Am. Heart J. 15: 232-234, Feb. 1938.

Pericarditis with effusion complicating myocardial infarction is infrequent *(6, 10)*. Master and Jaffe *(10)* reported that pericardial effusion secondary to myocardial infarction is very rare, perhaps even more so than the literature would tend to indicate. Hemopericardium when hemorrhagic disease is not an associated finding is seemingly unheard of in the situation under discussion. Wolff and Goldstein *(11)* reported 4 patients with hemorrhagic pericarditis associated with acute myocardial infarction who were treated with dicumarol. All were seen at autopsy over a 2-year period. They noted that these patients differed from all others in that they received dicumarol in addition to other treatment and at autopsy it was not possible to demonstrate rupture of the myocardium as the cause of pericardial hemorrhage. Since their report Wolff *(12)* has reported 5 additional patients. Leedham and Orbison *(13)* reported a patient with cardiac tamponade which developed in association with dicumarol therapy following myocardial damage with pericarditis secondary to nonpenetrating trauma of the heart. In an article dealing with the cause of hemopericardium Fulton *(14)* discussed the factors, including hemorrhagic diseases, which may be responsible. A hemorrhagic state, which resulted from therapeutic interference, though no disease state per se was detected, was described.

Although the fact that the pericardial tap yielded only 125 cc. of blood may seem to preclude the production of sufficient cardiac tamponade to precipitate the critical situation just reviewed, the factors in pericardial effusion which constitute a critical hazard are a function, not of the quantity of effusion alone, but of the speed of its formation *(5, 6, 15, 16)*. The pericardium does not readily distend and a relatively small amount of fluid suddenly entering the pericardial sac will produce a greater intrapericardial pressure than will a large amount of fluid entering over a long period of time *(17)*. How high a relatively small amount of fluid may raise the intrapericardial pressure was shown by Beck and Cushing *(18)* who were able to cause

(10) Master, A. M., and Jaffe, H. L.: Coronary artery thrombosis with pericardial effusion. J. A. M. A. 104: 1212-1214, Apr. 6, 1935.

(11) Wolff, L., and Goldstein, R.: Hemorrhagic pericarditis in acute myocardial infarction treated with dicumarol. Proceedings of the New England Cardiovascular Society, 43-45, 1949-1950.

(12) Wolff, L.: Personal communication.

(13) Leedham, C. L., and Orbison, J. A.: Cardiac tamponade associated with administration of dicumarol. Circulation 1: 1065-1072, 1950.

(14) Fulton, M. N.: Acute hemopericardium: its causes and clinical manifestations. M. Clin. North America 24: 1371-1386, Sept. 1940.

(15) Williamson, C. S., and Ets, H. N.: Rationale of therapeutic puncture in pericardial effusions; experimental study. Arch. Int. Med. 38: 206-216, Aug. 1926.

(16) Luisada, A. A.: Study of Cardiovascular Diseases. Williams & Wilkins Co., Baltimore, Md., 1948.

(17) Fishberg, A. M.: Heart Failure. 2d edition. Lea & Fiebiger, Philadelphia, Pa., 1950. p. 318.

(18) Beck, C. S., and Cushing, E. H.: Circulatory stasis of intrapericardial origin; clinical and surgical aspects of Pick syndrome. J. A. M. A. 102: 1543-1548, May 12, 1934.

an initial pressure of 21 cm. of water to drop to 0 cm. after 150 cc. of fluid were withdrawn. A very rapid effusion of from 150 to 250 cc. in hemopericardium or suppurative pericarditis can cause critical tamponade, but a slowly developing effusion of 1,000 cc. or more over a protracted period in tuberculosis may be tolerated *(16)*. An accumulation of 200 cc. of intrapericardial fluid has caused fatal tamponade of the heart *(17)*.

Severe congestive failure was present in this patient. Whether it was the cause or the effect of the pericardial effusion must be considered. A serous transudation into the pericardial cavity is the characteristic type of effusion found in congestive failure, and a common one as well *(16)*. The bloody fluid withdrawn from this patient was in no way characteristic of the physical properties of a transudate. The most reasonable explanation for his dramatic response, if it is assumed that congestive failure was singly responsible for the critical state, would be digitalization. The patient was given lanatoside C intravenously prior to the tap with no evident clinical improvement. His course continued downhill as manifested by increased distention of the cervical veins, decreasing urinary output, rising blood urea nitrogen, diminution of the erythrocyte count and hematocrit, cyanosis even in the oxygen tent, and the roentgenogram showing progression of congestion and pleural effusion bilaterally. The program of digitalization was interrupted 24 hours prior to the pericardial tap because of the increasing number of premature systoles. Resumption of digitalization was not undertaken until after the pericardial tap at a time when clinical improvement was already manifest. Therefore, it is reasonable to assume that the 125 cc. of bloody fluid were a significant contributing factor to the critical condition which developed. This is supported by (1) previous reports in the literature as to the possibility of relatively small amounts of effusion causing critical tamponade; (2) coincidental improvement of the patient almost immediately following the pericardial tap; (3) the presence of 9 percent of prothrombin activity; (4) failure of the patient to respond to previous intravenous injections of lanatoside C and mercaptomerin; (5) marked clinical improvement prior to full digitalization; and (6) maintenance of improvement with a rise in prothrombin activity to 58 percent at 0800 on 16 December.

The importance of congestive failure in promoting dicumarol toxicity has been pointed out by Powers *(19)* who was able to collect 5 patients who died of hemorrhagic complications following the administration of dicumarol. He reported that 2 of the 5 had in common a congestive heart failure which had been in effect for 7 weeks or longer. His interpretation was that chronic passive congestion of the liver interfered with the formation of prothrombin to such an extent that administration of what had previously been safe doses of dicumarol so severely

(19) Powers, J. S., Jr.: Toxicity of dicumarol; review of literature and report of 2 cases. Ann. Int. Med. 32: 146-152, Jan. 1950.

disrupted the clotting mechanism as to cause hemorrhage. Merrill *(20)* pointed out an associated factor in that diminished renal filtration due to congestive failure may cause enough retention of even small doses of dicumarol given by mouth to produce toxic levels. I believe that in this patient, and in several others seen in this and other hospitals, severe acute congestive heart failure may promote a similar effect.

CONCLUSIONS

Cardiac tamponade may result from the administration of anticoagulants to patients who have an associated myocardial infarction with pericarditis. The giving of anticoagulants to these patients should be recognized as hazardous. The importance of an accurate, detailed history is re-emphasized, with the added precaution that the history be carefully evaluated. Extreme caution should be observed when administering or planning to administer anticoagulants to patients with acute or chronic congestive heart failure.

FOLLOW-UP NOTE

Since the completion of this article the patient suddenly died about 2 months following the episode herein described. He had been symptom-free and seemed to be making good clinical progress. A single grave sign was noted in serial ECG's which showed persistent elevation of the RS-T segments, indicating poor healing or possibly aneurysmal formation at the site of the infarct. At necropsy the heart weighed 410 grams, was noted to be flabby and presented a wide area of hemorrhagic discoloration and softening of the posterior wall extending to the lateral wall of the left ventricle. This area was identified as an area of infarction which, on cross section, showed some fibrosis superimposed on the hemorrhagic necrosis of the muscle. Throughout the area of infarction there were sites of marked thinning but at no point was there any evidence of aneurysm or other abnormalities. The ostium of the left coronary artery showed a large arteriosclerotic plaque at the opening which reduced the lumen by 50 percent. The descending branch of the left coronary artery showed marked arteriosclerosis and calcification with marked reduction of the lumen. The circumflex branch was slightly affected by the arteriosclerosis. The right coronary artery showed a few arteriosclerotic plaques throughout and at a point 8.5 cm. from the origin there was a thrombus which completely occluded the vessel and which showed evidence of gradual slow occlusion and a fresh recent occlusion. The interpretation of these findings was that the preponderant involvement of the left coronary artery was the probable cause of the extensive infarction of the posterior wall in view of the finding of a distal lesion 8.5 cm. from the origin in the right coronary artery. The recent fresh occlusion in the right coronary artery was assumed to be the cause of death. An acute generalized pericarditis was also present.

(20) Merrill, A. J.: Edema and decreased renal blood flow in patients with chronic congestive heart failure; evidence of "forward failure" as primary cause of edema. J. Clin. Investigation 25: 389-400, May 1946.

Tuberculosis in the Northern Marianas

Shelton P. Sanford, *Captain, MC, U. S. N.*
Murdock S. Bowman, *Lieutenant, MC, U. S. N.*

THE island of Saipan and its 2 dependent islands, Tinian and Rota, contain over 95 percent of the natives of the northern Mariana Islands. They have an indigenous population of about 6,000 that is remarkably stable and relatively isolated. The population is not racially homogeneous. Most of the inhabitants are Chamorros, although there are over 1,000 Carolinians plus a scattering of Japanese, Koreans, and others. A tuberculosis hospital with a capacity of about 120 beds is maintained by the Civil Administration Unit authorities at Saipan. The center of medical treatment, except for leprosy, for the Northern Marianas is located on Saipan where about 80 percent of the natives live. For this reason, the group presents an unusual opportunity to observe the means of spread, natural history, and clinical course of tuberculosis under uniform conditions. It also affords an opportunity to compare the 2 larger racial groups, Chamorros and Carolinians. Much has been written about tuberculosis in the tropics, but relatively little material is available concerning the Northern Marianas. That this disease is prevalent in the native population of this hot, humid area is not surprising.

HISTORICAL BACKGROUND

Chamorros. In 1698 the Spanish defeated the Chamorros on Saipan and Tinian and transported them to Guam where smallpox and other epidemic diseases nearly exterminated them. During the next 150 years the addition of Filipino and Spanish settlers and smaller groups of other nationalities produced the racially-mixed Chamorro population of today. The first Chamorros returned to Saipan in 1818, but the principal influx seems to have come after 1880.

Carolinians. The Carolinians resettled Saipan in a series of waves of migration from the isolated central Carolines beginning in 1815. The first influx (about 200 people) asked permission to settle there because their atolls had been devastated by a typhoon. Another large group came in

1865, some of whom settled on Guam. The Guamanian colony moved to Saipan in 1901. Thus it can be seen that the Carolinians' period of contact with European and other races has been much shorter than that of the Chamorros.

TUBERCULOSIS IN MICRONESIA

The history of tuberculosis in the Marianas Islands is obscure. It cannot be said with certainty whether it was introduced by white explorers or by one of the many Polynesian, Chinese, or Japanese migrations into Micronesia. Doubtless, it has been reintroduced many times since its original appearance. The disease appears to have been prevalent in the Marianas before 1900 when Leach *(1)* reported cases from Guam.

Salecker *(2)*, using the von Pirquet skin test, reported that 55 percent of the Chamorros and 30 percent of the Carolinians reacted positively. More that 70 percent of a group of Carolinian immigrants from Woleai and Mogomog, however, reacted positively. Satterlee *(3)* listed yaws and tuberculosis as the most important diseases of Guam. From 1925 to 1929 the Japanese released statistics on the cases treated in government hospitals of all the mandated islands. The number of natives treated in 1 year ranged from 162 to 646. They reported in 1936 that they were providing special facilities for treating tuberculosis. The increase in the number of patients treated in Guam in the past 2 decades and in Saipan in the past 5 years probably reflects increased hospital facilities rather than increased morbidity.

From 20 September through 7 November 1949 a medical survey of the island of Saipan was conducted by the naval medical survey ship U. S. S. *Whidby (4)*. In a survey of 4,003 natives a general physical examination, a dental examination, and a photofluorographic examination were made, with follow-up roentgenograms of the chests of those suspected of having tuberculosis. Stool examinations were made on 34 percent of the persons examined and 86 percent were tested for tuberculosis with 0.0001 mg. of PPD tuberculin; 60 percent of those skin tests were positive. Based on roentgenographic findings 53 persons were found to have active pulmonary tuberculosis, 58 were suspected of having it, and in 22 the disease was arrested. About 2.7 percent of this population were considered to have tuberculosis in a contagious stage. Facilities for their care being available, all known patients with active or suspected pulmonary tuberculosis were brought into the hospital for clinical study. These patients, together with those

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(1) Leach, P.: Sanitary Report on Guam, L. I. (Aug. 7, 1899 to Dec. 31, 1899). Report of the Surgeon General, U. S. Navy, to the Secretary of the Navy, 1900. Government Printing Office, Washington, D. C., 1900. pp. 208-212.

(2) Salecker: Die Verbreitung der Tuberkulose auf den Marianan. Arch. f. Schiffs- u. Tropenhyg. 19: 369-376, 1915.

(3) Satterlee, R. C.: Sanitary Survey of the Island of Guam, 1928. Forwarded by the Governor Commandant of Guam, Aug. 28, 1928, to the Navy Dept., Washington, D. C.

(4) Lee, J. R.: Health and Sanitation Survey, Summary, Saipan Island, 1949.

already hospitalized, and follow-up surveys of their contacts provided the material for Lee's (4) study.

OBSERVATIONS

Table 1 shows the comparative populations of the Chamorros and Carolinians in the area under discussion. In studying the death rates in the various groups, 22 patients who died of proved tuberculosis in 1949 were used, with the rates calculated from the estimated midyear populations for each specified group. In patients with pulmonary tuberculosis not proved by autopsy, the diagnostic criteria used by the Trudeau Sanatorium were followed. The death rate for all groups in this

TABLE 1. *Estimated midyear population by age and sex of Chamorros and Carolinians on Saipan, Tinian, and Rota, 1949*

	Age (years)*		Total
	Under 15 years	15 years and over	
Chamorro	2568	2203	4771
Male	1208	1125	2333
Female	1360	1078	2438
Carolinian	422	716	1138
Male	200	266	466
Female	222	450	672
Total	2990	2919	5909

*These age groups were used because population statistics were available only in these categories.

area (372.3 per 100,000 population as shown in table 2) was more than 12 times that for the United States in 1948 (5). The Carolinian death rate was more than 6 times that for the Chamorros. The rates of 2,000 and over for Carolinian males are astronomical and represent more than 1 death in every 50 Carolinian males in 1949.

In the 15-month period from 1 January 1949 through 31 March 1950, 34 Chamorros and Carolinians died of proved tuberculosis. Since late in 1944 facilities have been available there for the hospitalization of tuberculous patients. Because serial roentgenograms were available during this period, it was decided to use these and the clinical histories to get an index as to the severity and duration of the disease in this area. From tables 3 and 4, in which the cases were broken down ac-

(5) Guralnick, L., and Glaser, S.: Tuberculosis mortality in United States, 1948. Pub. Health Rep. 65: 468-493, Apr. 7, 1950.

TABLE 2. *Number of deaths and death rates per 100,000 for tuberculosis (all forms) by age and sex for Chamorros and Carolinians inhabiting Saipan, Tinian, and Rota, 1949*

	Age (years)					
	Total		Under 15		15 and over	
	Number	Rate	Number	Rate	Number	Rate
Chamorro	9	188.6	6	233.6	3	136.2
Male	4	171.5	4	331.1	0	0
Female	5	205.1	2	147.1	3	278.3
Carolinian	13	1142.4	4	947.9	9	1256.9
Male	10	2145.9	4	2000	6	2255.6
Female	3	446.4	0	0	3	666.7
Total	22	372.3	10	334.4	12	411.1

cording to race and sex, and then by age, it was striking to note that 41.2 percent died in less than 6 months from the onset; 67.7 percent in less than 1 year; and 29 percent were in children 4 years of age and under. More deaths occurred in males than females and more in Carolinians than Chamorros, in spite of the preponderant number of Chamorros. There were strikingly few deaths in the older age groups. These facts seem to indicate that the disease here is, indeed, malignant, particularly in children. Also, the Carolinians are apparently more susceptible to the rapidly progressing forms of the disease than are the Chamorros.

In the 15-month period previously mentioned, 169 patients with proved tuberculosis were brought under clinical observation. Those with active tuberculosis were retained for treatment. All those admitted had a roentgenogram of the chest, multiple sputum examinations, sedimentation rate, stool examination for ova and parasites, urinalysis, complete blood count, and history and physical examination. Patients with questionably active tuberculosis were hospitalized for about 1 month, at the end of which time the tests were repeated. Those whose disease was considered inactive were discharged to the clinic where periodic examinations were made. Tuberculosis of the respiratory system comprised 95.9 percent of the patients (table 5). Lack of adequate facilities and personnel accounts for the apparent low rate of nonrespiratory tuberculosis, and it is probable that many others were not found. The diagnoses were proved in the case of those listed. All of the patients with disseminated tuberculosis and tuberculous meningitis were proved by autopsy. Tuberculous pericarditis was a prominent form of nonrespiratory tuberculosis, being found exclusively in children under 3 years of age. A number of the patients with respiratory tuberculosis were

TABLE 3. *Duration of disease in 34 Chamorros and Carolinians dying of tuberculosis (all forms) from Saipan, Tinian, and Rota, from 1 January 1949 through 31 March 1950, by race and sex*

	Duration												Total
	Less than 6 mo.		6 mo. - 1 yr.		1 - 2 yr.		2 - 3 yr.		3 - 4 yr.		More than 4 yr.		
	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	
Chamorro	6	17.6	4	11.8	2	5.9	1	2.9	1	2.9	1	2.9	15
Male	3	8.8	1	2.9	1	2.9	0	0	0	0	0	0	5
Female	3	8.8	3	8.8	1	2.9	1	2.9	1	2.9	1	2.9	10
Carolinian	8	23.5	5	14.7	1	2.9	2	5.9	2	5.9	1	2.9	19
Male	7	20.6	3	8.8	1	2.9	1	2.9	1	2.9	1	2.9	14
Female	1	2.9	2	5.9	0	0	1	2.9	1	2.9	0	0	5
Total	14	41.2	9	26.5	3	8.8	3	8.8	3	8.8	2	5.9	34

*Of total.

TABLE 4. *Total duration of disease in 34 Chamorros and Carolinians dying of tuberculosis (all forms) from Saipan, Tinian, and Rota, from 1 January 1949 through 31 March 1950, by age*

Age (years)	Duration												Total
	Less than 6 mo.		6 mo. - 1 yr.		1 - 2 yr.		2 - 3 yr.		3 - 4 yr.		More than 4 yr.		
	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	
Under 1	2	5.9	0	0	0	0	0	0	0	0	0	0	2
1 - 4	8	23.5	2	5.9	0	0	0	0	0	0	0	0	10
5 - 14	1	2.9	1	2.9	1	2.9	0	0	0	0	0	0	3
15 - 24	2	5.9	0	0	0	0	2	5.9	2	5.9	0	0	6
25 - 34	0	0	1	2.9	0	0	0	0	1	2.9	1	2.9	3
35 - 44	1	2.9	2	5.9	0	0	0	0	0	0	0	0	3
45 - 54	0	0	2	5.9	1	2.9	1	2.9	0	0	0	0	4
55 - 64	0	0	1	2.9	0	0	0	0	0	0	1	2.9	2
65 and over	0	0	0	0	1	2.9	0	0	0	0	0	0	1
Total	14	41.2	9	26.5	3	8.8	3	8.8	3	8.8	2	5.9	34

*Of total.

found or proved only at autopsy. These were patients who died at home or who died shortly after admission before an adequate clinical workup could establish the diagnosis.

In the distribution of all forms of tuberculosis (table 6), 28.4 percent occurred among the Carolinians, although they comprised only about 19 percent of the population of the 2 groups. More patients were female than male, in spite of the fact that only 40 percent of the group were female. The largest number in any of the age groups among the Carolinians was in the group 1 to 4 years of age and most of these were patients with rapidly advancing progressive disease.

Table 7 shows the distribution of 162 patients with pulmonary tuberculosis by clinical classification according to the National Tubercu-

TABLE 5. *Number of patients and percents by specified form of tuberculosis in the Chamorros and Carolinians of Saipan, Tinian, and Rota, 1 January 1949 through 31 March 1950*

	Number of patients*	Percent of total
Tuberculosis (all forms)	169	100
Tuberculosis of the respiratory system	162	95.9
Tuberculosis, other forms	28	16.6
Tuberculosis of the meninges and central nervous system	3	1.8
Tuberculosis of intestines and peritoneum	1	.6
Tuberculosis of the bones and joints	2	1.2
Tuberculosis of the skin	0	0
Tuberculosis of the lymphatics (except bronchial and mediastinal)	7	4.1
Tuberculosis of the genitourinary tract	3	1.8
Tuberculosis of the pericardium	9	5.3
Disseminated tuberculosis	4	2.4

*Many patients had more than one type of tuberculosis.

losis Association. Moderately advanced cases were most frequent. Twenty-six percent of the patients with primary tuberculosis died, whereas only 12.9 percent of those with the reinfection type died. The former was apparently more fatal. We know that many cases of primary tuberculosis are probably passed off as severe colds. The same might be thought to be true here except that in the period of time of this study over 90 percent of the population were x-rayed.

DISCUSSION

Tuberculosis in the tropics has high morbidity and mortality rates and often runs a rapidly fatal course. In fact it sometimes has the characteristics of an epidemic. These features are often attributed to (1) the racial susceptibility of "nonwhites," (2) insanitary and crowded living conditions, and (3) lack of facilities for the segregation of those

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TABLE 6. *Distribution of 169 patients with tuberculosis (all forms) among the Chamorros and Carolinians of Saipan, Tinian, and Rota, 1 January 1949 through 31 March 1950, by age and sex*

	Total		Age (years)																	
			Under 1		1 - 4		5 - 14		15 - 24		25 - 34		35 - 44		45 - 54		55 - 64		65 and over	
	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*	Number	Percent*
Chamorro..	121	71.6	4	2.4	11	6.5	20	11.8	20	11.8	16	9.5	15	8.9	18	10.7	12	7.1	5	3.0
Male.....	48	28.4	3	1.8	6	3.5	11	6.5	4	2.4	4	2.4	9	5.3	6	3.5	2	1.2	3	1.8
Female....	73	43.2	1	.6	5	3.0	9	5.3	16	9.5	12	7.1	6	3.5	12	7.1	10	5.9	2	1.2
Carolinian	48	28.4	0	0	11	6.5	5	3.0	6	3.5	5	3.0	7	4.1	7	4.1	5	3.0	2	1.2
Male.....	30	17.7	0	0	9	5.3	2	1.2	2	1.2	1	.6	6	3.5	5	3.0	4	2.4	1	.6
Female...	18	10.7	0	0	2	1.2	3	1.8	4	2.4	4	2.4	1	.6	2	1.2	1	.6	1	.6
Total	169	100.	4	2.4	22	13.0	25	14.8	26	15.3	21	12.4	22	13.0	25	14.8	17	10.1	7	4.1

*Of total.

TABLE 7. *Number of cases and deaths from pulmonary tuberculosis by type* among the Chamorros and Carolinians of Saipan, Tinian, and Rota 1 January 1949 through 31 March 1950*

Classifications	Number of patients		
	Living	Dead	Total
Primary	34	12	46
Reinfection	101	15	116
Minimal	32	0	32
Moderately advanced	50	1	51
Far advanced	19	14	33
Total	135	27	162

*National Tuberculosis Association classification.

suffering from the disease. The first and second of these factors applied to the Chamorros and Carolinians. Today tuberculosis is regarded as the chief cause of death in the Philippines, Jamaica, many parts of Africa, and, probably, in the East Indies. It is the most important single cause of death in Micronesia.

In tropical regions the natives living near sea level suffer more than those living in the mountain plateaus and where the humidity is high rather than in arid sections. The disease gains headway in the tropics in the rainy season and diminishes in prevalence during the dry season. Saward et al. *(6)* carried out important studies on the spread of tuberculosis in the Negroes of Jamaica. They found that tuberculosis in these Jamaicans pursues a much more rapid course than that in white people of European extraction and is readily transferred from a person with the disease to other members of the household, adults as well as children, so that many of them may die of tuberculosis within 2 years after it has been introduced into the family. This fact was also observed on Saipan when a roentgenographic survey of a Carolinian family group in which there had been 3 deaths within 6 months revealed 7 more active cases in the remaining 22 members of the group.

The source of infection was often difficult to determine, but when the disease was found, it usually occurred in more than 2 members of the family unit. This was particularly true among the Carolinians. In this group, a fundamental feature of their social and economic organization is the co-operative activity carried on by a large circle of relatives comprising several closely related families. Within this large related group there is much co-operative labor, interfamily visiting, and close social contact. Unfortunately, although this co-operative char-

(6) Saward, E. J., et al.: Studies on Tuberculosis. American Journal of Hygiene Monograph Series, No. 16. Johns Hopkins Press, Baltimore., Md., 1941.

acteristic is a source of strength to the community, it also facilitates the spread of tuberculosis. Among Carolinians a sequence of events frequently occurred in the following manner. Patient A is admitted to the hospital with an easily-diagnosed, far-advanced case of pulmonary tuberculosis. The patient's family is x-rayed and one or more patients with active disease are found and hospitalized. Patient A is in the terminal stage and dies within a few days. Suddenly the medical officer is confronted with one or more patients who have been on the tuberculosis wards for some time and are asking for permission to attend the funeral of patient A because they are part of the family. Confused because their names are different, and because he thought all of the members of the family had been x-rayed, the medical officer finds that the "family" consists of not only parents and their progeny, but of many other relatives as well, all living interchangeably in two or more houses. The scope of intimate contact is thus enlarged. It is therefore necessary to individualize each case and to discover who were actually part of a certain family and who were not.

It is difficult to see how bovine tuberculosis could play any part in the spread of the disease on Saipan because the cattle are used almost exclusively for labor. Very few are slaughtered for beef and the natives rarely drink cow's milk. Furthermore, the viscera of all cattle slaughtered for beef are examined grossly and we have seen nothing in them even suggestive of tuberculosis.

The high incidence of infestation with intestinal parasites, frequently heavy and often multiple in the native population, was believed to be a contributing factor in the high incidence of tuberculosis because of the accompanying poorer nutrition and weakened resistance. Roundworms and hookworms were the chief offenders, although *Strongyloides stercoralis, Giardia lamblia,* and *Trichuris trichiura* were quite common. Because tuberculosis and intestinal parasites thrive under insanitary conditions, poorer sanitation combined with heavy intestinal parasitism were believed to be important factors in the higher incidence and mortality among the Carolinians than among the Chamorros and in the children of both groups. Poorer personal hygiene, more crowding in the homes, and poorer sanitary facilities were found among the Carolinians than among the Chamorros. These factors promote more contact as well as more chance of overwhelming infections. The fact that the Carolinians have not been subjected to contact with Europeans as long as the Chamorros or to the same extent may account for less racial resistance to tuberculosis in this group. The Carolinians also tend to live in more isolated areas and in segregated groups farther away from dispensary facilities. They are very shy and frequently will not come to see a doctor until they are extremely ill. We believe that these factors explain the difference in tuberculosis rates in the two groups. Why the highest death rate, out of all proportion, occurred in the Carolinian males is unknown.

In studying the disease clinically, a discrepancy was frequently found between the severity of the disease as indicated by the roentgenogram of the chest and the constitutional reaction. A normal temperature curve often accompanied a moderate to far-advanced infection. Because increased temperature is the normal body response to infection, here was a further indication of a lack of resistance to the disease.

SUMMARY

Tuberculosis is the chief health problem among the people of the northern Marianas. There is a far higher mortality among the Carolinians than among the Chamorros, this probably being due in part to their racial background and in part to their poorer living conditions. Children from both groups suffer a high mortality from the disease. The high incidence of intestinal parasitic infestations and the generally poor dietary standards are believed to contribute to the incidence of tuberculosis in these people. The disease in the Marianas often runs a rapidly fatal course, showing comparatively little tendency to become arrested. Bovine tuberculosis is conspicuous by its absence. The constitutional symptoms from the disease are often slight or absent, even when the roentgenogram indicates moderately to far-advanced disease. The "negative" roentgenograms were usually remarkably clear, showing no evidence of histoplasmosis. It is recommended that when some of the presently uninhabited islands in the northern Marianas are settled again, the groups moving in be thoroughly screened for tuberculosis so as to limit its spread to other areas.

BOOK REVIEWS

Manic-Depressive Psychosis and Allied Conditions, by *Leopold Bellak, M. D.,* Clinical Assistant Professor of Psychiatry, New York Medical College, Fifth Avenue and Flower Hospitals; with *Blaise Pasquarelli,* M. D., Research Associate, New York State Psychiatric Institute; *Ernest Parkes,* M. A., Instructor in Psychology, New York University; *Sonya Sorel Bellak,* and the collaboration of *Sydell Braverman,* M. A. Foreword by *Winfred Overholser,* M. D. 306 pages. Grune & Stratton, Inc., New York, N. Y., publisher, 1952. Price $9.75.

Following the publication in 1948 of his volume on dementia praecox, Dr. Bellak undertook the job of reviewing the literature on manic-depressive psychosis. This book reviews about 1,200 references. Its purpose is not to judge the validity of the material reviewed, but rather to collect under one cover the views of many workers in this field.

In the introduction, the author presents his own formulations of manic-depressive psychosis. He writes of a "multiple-factor psychosomatic theory of manic-depressive psychosis." He believes "that the etiologic

factors of this syndrome may range from a hypothetically almost completely psychogenic nature to a hypothetically almost completely organic nature." He does not expound on what such organic factors might be. With respect to the psychogenic factors, he is more explicit. He believes that a weakening of the ego is involved and that manic-depressive psychosis represents an equilibrium established in a personality structure essentially fixed at oral levels (after Abraham) which suffers from a decrease in ego strength. He postulates that the ego strength in this syndrome is greater than in schizophrenia and less than that found in obsessional neurosis.

The remainder of the book deals exclusively with a review of the literature. Chapter 1 deals with definition and description. Here Dr. Bellak, in common with other workers, is unable to find universal agreement either etiologically or dynamically on just what, exactly, is meant by the term manic-depressive psychosis. Even descriptively there is disagreement in this area. In the chapter on etiology, data is presented on psychogenic theories and on constitutional, neurologic, biochemical, hereditary, and precipitating factors. In the chapter on physiopathologic studies the work on carbohydrate metabolism, biochemical, endocrine, and neurologic studies is reviewed. Other chapters deal with vital statistics on psychopathologic studies, diagnosis and symptomatology, manic-depressive psychosis in childhood, treatment (with electric shock, metrazol, insulin, psychotherapy, and psychosurgery), complications and sequelas, prognosis, and prevention.

The third portion of the book, devoted to a review of allied conditions, contains chapters on psychotic reactions to pregnancy, involutional psychoses, reactive depressions, and depressions of old age and suicide. The latter chapter points up the fact that suicide is a general psychiatric problem not limited to manic-depressive psychosis.

The author has succeeded in condensing into one volume a mass of research and writings on manic-depressive psychosis and allied conditions. Those interested in research in these areas will find the book a valuable reference source. Psychiatrists preparing for board examinations may also find it useful.—*Commander M. Shoor, MC, U. S. N.*

Principles and Practice of Aviation Medicine, by *Harry G. Armstrong,* M. D., F. A. C. P., The Surgeon General, United States Air Force. 3d edition. 476 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publisher, 1952. Price $7.50.

This book has undergone thorough revision and the addition of new material has brought the work as nearly up to date as possible. The book is of value not only as a comprehensive text, but also as a book of reference and should be in the hands of every physician who deals with the medical aspects and problems of aviation.

—*Rear Adm. B. Groesbeck, Jr., MC, U. S. N.*

Malignant Degeneration in Multiple Neurofibromatosis[1]

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MALIGNANT degeneration as a complication of von Recklinghausen's disease is quite rare. About 88 cases in the literature, not including the one we are describing have been reported (2). Multiple neurofibromatosis in itself is not an uncommon condition and is estimated to occur in about 1 in 2,000 persons. It has been reported in all races (3).

Etiology. The cause of this condition is unknown, but there appears to be a close connection between the appearance or exacerbation of this disease and various endocrine disturbances. It also appears to be hereditary and is commonly associated with other congenital anomalies.

Symptoms and signs. All types of nerves, including those of the sympathetic and parasympathetic systems, may be involved. As a rule there are no symptoms but occasionally a tumor will require removal because of pressure on an adjacent nerve. This is found most commonly in tumors arising from the intercostal nerves or their branches. These tumors may make their appearance during infancy or early childhood but most commonly are recognized during puberty (2). Pigmentation of the skin ("cafe-au-lait" spots) is a very common finding associated with this disease and was a characteristic finding in all of the 6 patients with multiple neurofibromas observed in this hospital in the past 2 years. The size of the subcutaneous nodules may vary from that of a hen's egg to that of a small match head. One patient recently observed in this hospital had multiple tumors the size of

(1) 118th Station Hospital.
(2) Herrmann, J.: Sarcomatous transformation in multiple neurofibromatosis (von Recklinghausen's disease); report of 4 cases. Ann. Surg. 131: 206-217, Feb. 1950.
(3) Sharpe, J. C., and Young, R. H.: Recklinghausen's neurofibromatosis; clinical manifestations in 31 cases. Arch. Int. Med. 59: 299-328, Feb. 1937.

a hen's egg in the subcutaneous tissue of the neck, both axillas, the chest, the abdomen, and the arms. These tumors may occur anywhere in the body. The usual sites of metastasis for this type of tumor, if malignant, are the lungs, but as a rule metastases are infrequent and occur late in the course of the disease.

Treatment. Caution should be used in deciding to remove one of these growths or cafe-au-lait spots. Some observers believe that incomplete removal of such a tumor or local trauma is the stimulating factor in causing malignant degeneration. Malignant transformation may, however, take place in parts of the body distant from the tumor or spot excised. This may well have occurred in the case reported here.

CASE REPORT

A 29-year-old soldier entered this hospital on 17 February 1950 complaining of abdominal pain of 8 days' duration just to the left of the umbilicus. In 1947 he first became aware that he had multiple neurofibromas. At that time one small cafe-au-lait spot and one neurofibroma was removed from the subcutaneous tissue of his abdomen. His twin brother and his father are living and well and apparently do not have von Recklinghausen's disease. His mother is 53 years old and has the disease.

On 15 December 1949 the patient was on maneuvers and was exposed to cold, damp weather for about 7 weeks. He returned to his camp on 9 February 1950, stating that during the preceding 3 weeks he had felt weak and rundown. On the day of his return to camp he had lunch at 1130 hours and 40 minutes later began to have sudden, moderately sharp pains to the left of the umbilicus which gradually radiated to the right of the umbilicus. These pains would last about 5 minutes and then disappear for from 15 to 20 minutes. The pains continued intermittently for the next 6 hours and gradually became worse at which time the patient had a moderate feeling of chilliness and vomited 3 times within 30 minutes. The chilliness lasted for about 90 minutes. At that time the patient was examined at the local dispensary where his temperature was found to be 103° F. and a tentative diagnosis of acute gastroenteritis was made.

He was kept in the dispensary for 7 days during which time he received sulfonamides with no relief of symptoms. He had no more vomiting or chills. During his stay in the dispensary occult blood was found in his stools. His erythrocyte count was 2,800,000. His hemoglobin was 7.8 grams per 100 cc. His hematocrit was 31 and his sedimentation rate was 47. He was transferred to this hospital on 17 February with a tentative diagnosis of gastrointestinal bleeding and hypochromic anemia. On admission his temperature was 100.4° F.; his pulse was 120; and his blood pressure was 150/80. He appeared chronically ill. His skin had a lemon-yellow hue. No petechias were noted. There were multiple small discrete nodules in the subcutaneous

tissues throughout his body. There were at least 3 typical cafe-au-lait spots, the most prominent of which was on his left shoulder. This lesion measured about 2 cm. in diameter. There was no generalized lymphadenopathy. Examination of the abdomen revealed slight distention with tenderness and some guarding in his left rectus abdominis muscle. There was slight rebound tenderness on his left side and an ill-defined mass about the size of a lemon was palpable about 5 cm. to the left of his umbilicus. His spleen was easily palpable about 3 fingerbreadths below the costal margin. Bowel sounds were active. Two days following admission the patient complained of increased pain in the left side of his abdomen. His temperature was 100° F. and his pulse was 120. There was slight abdominal distention and a definite tumor mass was easily palpable just to the left of his umbilicus. Definite rebound tenderness was elicited throughout his abdomen but was most marked over the mass. It was believed at that time that he had an intraperitoneal abscess the exact nature of which was unknown and intestinal decompression with a Miller-Abbott tube was started. Antibiotics, intravenous fluids, and 500 cc. of whole blood were given.

On the third hospital day his temperature was 102° F., his pulse was 100, his urinary output was adequate and his general condition was unchanged. The Miller-Abbott tube was well down in his small bowel. On the fourth hospital day his temperature was normal, his abdominal pain had practically disappeared, and he felt better. On this day there was no tenderness to palpation and no rebound tenderness was elicited. His spleen remained enlarged. His hemoglobin was 10 grams per 100 cc. with a 4.4 million erythrocyte count. His bleeding time, clotting time, and platelet counts were within normal limits. His nonprotein nitrogen was 39 mg. per 100 cc. His plasma chlorides were 90 mEq per liter. His icterus index was 15 with a normal total serum bilirubin. On the fifth hospital day his temperature was 98.8° F. and he felt much better.

From that time on his general course was one of definite improvement with almost complete resolution of the tender mass palpable in the abdomen. A roentgenogram of the abdomen, however, revealed an irregular mass opposite the third lumbar vertebra to the left of the midline. The enlarged spleen was also seen on this film. By the ninth hospital day the patient had received 2,500 cc. of whole blood and his general condition was very satisfactory. He remained afebrile and except for occasional pain in the left side of his abdomen over the formerly palpable mass, he was without complaint. He was then transferred from the medical to the surgical service and on the seventeenth hospital day a laparotomy was performed. Preoperatively it was thought that this patient probably had a retroperitoneal lymphosarcoma and that the mass in his abdomen was probably a necrotic lymph node which had ruptured and extravasated some of its necrotic contents into his peritoneal cavity. This diagnosis was favored by the continued anemia and the patient's cachectic appearance on admission.

At operation a generalized inoperable sarcomatous process was found in the preaortic and mesenteric areas. This process consisted of multiple, symmetrical tumors from 3 to 4 cm. in diameter. The previously palpable mass within the abdomen consisted of 2 lobular necrotic tumors each about the size of a hen's egg, situated in the mesentery just proximal to the mesenteric border of the jejunum over which the omentum had become securely attached. The tumor had not invaded the wall of the jejunum. A biopsy specimen was taken to establish the type of sarcoma present and the abdomen was closed.

Microscopically the specimen consisted of interlacing bundles of connective tissue with spindle-shaped nuclei and numerous dilated vascular channels. Most of the nuclei were uniform in size and only rarely was a mitotic figure seen. Many cells were vacuolated. A collagenic capsule was irregularly infiltrated by these cells and focal areas of necrosis and acute inflammatory reaction were present. A diagnosis of neurofibrosarcoma of the mesentery was made. The postoperative course was smooth and the patient was transferred to Brooke Army Hospital, Fort Sam Houston, Tex., for radiation therapy.

SUMMARY

About 88 patients with malignant degeneration in multiple neurofibromatosis have been reported. To this number we have added 1 case. This patient presented preoperative signs and symptoms of intraperitoneal abscess. Most of the patients referred to were reported prior to 1910 so that this condition is probably more common than statistics would indicate. Biopsy specimens are not necessary to make a diagnosis in the usual case of von Recklinghausen's disease. This procedure is usually contraindicated because of the probability of initiating malignant degeneration, either locally or in some distant neurofibroma.

Lesions of the Pericardium

A Review of One Hundred Cases (1)

Philip G. Keil, *Major, U. S. A. F. (MC)* (2)

Donald J. Schissel, *M. D.* (3)

LESIONS of the pericardium are frequently first recognized by the prosector. This fact prompted a review of our cases. The study, in addition to its clinical implications, entailed examining about 15,000 clinical records and autopsy protocols covering 3½ years. The records and protocols are those of a 500-bed general hospital located in the Midwest. The patient population is adult and 98 percent male. Significant pericardial disease was found in 100 cases (table 1).

LESIONS SECONDARY TO MALIGNANCY

Of the 30 patients with malignant infiltration or metastases, 20 were secondary to bronchogenic carcinoma, 3 to malignant melanoma, 2 to myelogenous leukemia, 2 to carcinoma of the thyroid, 2 to carcinoma of the stomach, and 1 to carcinoma of the larynx. Pericardial involvement was discovered by postmortem examination in 27 and by exploration in 3. None was detected clinically. The ECG was not of much help although in 2 patients there were changes compatible with cardiac involvement. All the patients' symptoms and clinical findings were attributed to the underlying condition. We regard the development of persistent tachycardia not explained by fever suggestive of extension to the heart. The development of minor electrocardiographic changes such as diminishing QRS voltage and loss of T-wave voltage or slight T-wave inversion is also suggestive.

Scott and Garvin (4) found metastatic lesions involving the heart or pericardium in 118 of a series of 1,082 cases of deaths due to malignant disease. Carcinoma of the bronchus and breast accounted for 48

(1) Sponsored by the Veterans Administration with the approval of the Chief Medical Director. Statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

(2) U. S. Air Force Hospital, Dow Air Force Base, Bangor, Maine.

(3) Veterans Administration Hospital, Des Moines, Iowa.

(4) Scott, R. W., and Garvin, C. F.: Tumors of heart and pericardium. Am. Heart J. 17: 431-436, Apr. 1939.

percent of this series. In nearly all cases this involvement was by extension. They believed that development of congestive failure without other apparent cause was the most important clinical finding indicating cardiac metastases. One of our patients, a 21-year-old man, had pericardial and myocardial involvement at the time of thoracotomy and subsequently died of heart failure. Involvement of the pericardium was by extension in our cases.

TABLE 1. *Lesions of the pericardium*

Malignancy	30
Undetermined cause	17
Rheumatic fever	14
Uremia	11
Myocardial infarction	10
Secondary to pneumonia	4
Disseminated lupus erythematosus	4
Tuberculosis	4
Cyst	1
Secondary to subdiaphragmatic abscess	1
Traumatic	1
Rupture of aorta	1
Polyserositis	1
Irradiation	1
Total	100

In 5 patients neoplastic infiltration involved the pericardium and the myocardium. Two had fulminating myelogenous leukemia and 3 had widely invasive melanomas. In none was pericardial involvement suspected clinically, though 1 had suggestive electrocardiographic changes. The 2 patients with myelogenous leukemia showing extensive invasion of all visceral structures had terminal cardiac enlargement and pulmonary infiltration. In the other 5 patients (2 with thyroid, 2 with gastric, and 1 with laryngeal carcinoma) the pericardium was involved by simple extension and the condition was not suspected clinically.

LESIONS SECONDARY TO RHEUMATIC FEVER

Of the 14 patients whose pericardial involvement was due to rheumatic fever, the disease was fibrinous in 8 and fibrous in 6. The 6 were proved by autopsy. All had dense fibrous pericarditis with obliteration of the pericardial sac and an associated endocarditis and myocarditis. Six of the 8 patients with acute rheumatic pericardial disease were discovered clinically. All recovered. Two, discovered at necropsy, had rheumatic pancarditis and died of myocardial failure. Of the 8 patients with acute pericarditis, 4 had severe, constant, nonradiating retrosternal pain and 4 had dyspnea. One had no symptoms and another had only migratory polyarthritis. Only 2 had friction rubs and developed effusions. Six had electrocardiographic changes typical

of pericarditis (5-8). Characteristically, electrocardiographic changes developed from 10 to 14 days after the onset of the infection and returned to normal a fortnight after the infection subsided. The ECG appears to be the most reliable criterion of pericardial involvement in this type of pericarditis. Three patients of this group are of special interest. One, a 54-year-old man, gave a history of mild cardiac disability since his youth when he was forced to drop out of long marches because of dyspnea. When seen 4 years prior to death he had a huge heart and severe congestive failure. Angiocardiography, done 18 months before his death, showed a massive pericardial effusion, yet he responded to therapy and was discharged. When he returned in January 1950, a review of the films showed that massive pericardial effusion had been present since early in 1946. Pericardiotomy was performed but he died later of tamponade. Autopsy revealed a tetralogy of Fallot with rheumatic endocarditis, myocarditis, and pericarditis.

Two patients, aged 22 and 29 years, were almost identical clinically. Neither was definitely diagnosed, although rheumatic fever was suspected in 1. On admission both were extremely ill with a high fever and cardiac failure but without murmurs or friction rubs. Both had cardiac enlargement and electrocardiographic changes, 1 typical of and 1 suggestive of pericarditis. The sedimentation rate was 1 mm. in 1 hour in the 29-year-old patient. Both had leukocytosis and died after a short period of hospitalization. Autopsy showed rheumatic pancarditis, congestive failure, perihepatitis, and perisplenitis.

LESIONS SECONDARY TO UREMIA

The pericarditis of uremia is ordinarily considered to be a late complication and its cause is not known. Effusion is unusual and the diagnosis is frequently made post mortem. Of the 11 patients with pericarditis of uremic origin, a pericardial friction rub was heard in 5, and in those 5 the diagnosis was clinical. In the others there was no symptom or sign of significance. The ECG was helpful in 1, a 25-year-old Negro with peritonitis following appendiceal perforation. He was extremely ill, septic, and developed oliguria and azotemia followed by left ventricular failure. A friction rub became audible and the ECG was characteristic. After a protracted and stormy course during which he was given antibiotics and supportive cardiac therapy, he recovered and

(5) Fowler, W. M.; Rathe, H. W.; and Smith, F. M.: Electrocardiographic changes following ligation of small branches of coronary arteries. Am. Heart J. 8: 370-387, Feb. 1933.

(6) Vander Veer, J. B., and Norris, R. F.: Electrocardiographic changes in acute pericarditis; clinical and pathological study. Am. Heart J. 14: 31-50, July 1937.

(7) Bellet, S., and McMillan, T. M.: Electrocardiographic patterns in acute pericarditis; evolution, causes and diagnostic significance of patterns of limb and chest leads; study of 57 cases. Arch. Int. Med. 61: 381-400, Mar. 1938.

(8) Nay, R. M.: Electrocardiogram in acute pericarditis. J. Indiana M. A. 42: 222-228, Mar. 1949.

2 years later had no evident cardiac abnormality. The others died and autopsy revealed the pericardial changes associated with renal failure.

LESIONS SECONDARY TO MYOCARDIAL INFARCTION

All of the patients with pericarditis associated with myocardial infarction were diagnosed at autopsy. All showed the usual fibrinous or fibrous pericarditis and, if the infarction were recent, from 25 to 150 cc. of serosanguineous fluid was present. Because electrocardiographic changes due to myocardial injury outweigh changes caused by localized pericardial involvement and an effusion of a magnitude recognizable on physical examination or the roentgenogram is rarely seen, the diagnosis must hinge on hearing a friction rub. The rub occurs in only a small percent of cases and may be transient and of low intensity *(9)*.

LESIONS SECONDARY TO PNEUMONIA

Of the 4 patients with pericarditis complicating pneumonia, 2 died and 2 recovered. One who died had a recurrence of bronchogenic carcinoma in the left bronchial stump 1 year after pulmonary resection. The stump had perforated, resulting in a bronchopleural fistula, empyema, and pericarditis. Postmortem examination showed no evidence of neoplastic invasion of the pericardium. The pericardial sac contained 750 cc. of purulent exudate. The other patient who died had bilateral lobar pneumonia with infected pleural spaces and an early terminal purulent pericarditis which may have been of septicemic origin. Neither had electrocardiographic changes.

The other 2 patients are of interest because they apparently had pericarditis with pneumonia, but did not develop a discernible purulent exudate in the pericardial sac or the classical lobar pneumonia and empyema on the left side and later pericarditis. This development could well have been aborted by antibiotic therapy. One patient entered the hospital to convalesce after the diagnosis had been made elsewhere on the basis of clinical course, roentgenographic findings, and a pericardial friction rub. There was no evidence of a rheumatic or other cause. The ECG's were still showing evolutionary changes, but soon returned to normal. The other patient was extremely ill with pneumonia in the lower lobe of the left lung. During the height of the illness, he developed severe precordial pain, tachycardia, dyspnea, and a harsh friction rub. He recovered completely without residuals. The ECG showed changes typical of pericarditis. Purulent pericarditis complicating penumonia will probably become a rarity in the future because of the effectiveness of antibiotic therapy. The clinical diagnosis in these patients is not difficult and should always be considered in patients with pneumonia in the lower lobe of the left lung associated with empyema.

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(9) Levine, S. A.: Clinical Heart Disease. 3d edition. W. B. Saunders Co., Philadelphia, Pa., 1945. p. 110.

LESIONS SECONDARY TO TUBERCULOSIS

In all of the patients with tuberculosis of the pericardium, the diagnosis was proved by portmortem examination. Two had the complication as a terminal event of far-advanced bilateral pulmonary disease. Both had a granular shaggy pericarditis, 1 without fluid and the other with 175 cc. of seropurulent exudate. In the patient with fluid, the heart tones prior to death were distant and the ECG showed low voltage. In neither was a friction rub heard. The third patient had disseminated tuberculosis and miliary implants on the pericardium. He complained of severe precordial pain and dyspnea for 10 days before his death, but no friction rub was noted. The fourth patient was admitted with a history of cough, anterior chest pain, weight loss, chills, and fever of several months' duration. The roentgenogram showed a right hilar mass considered to be bronchogenic carcinoma. Narrowing and displacement of the right main stem bronchus were demonstrated bronchoscopically. The aspirated bronchial secretions suggested malignancy. An exploratory thoracotomy was performed and the entire mediastinum was found to be fixed. The surgeon believed he was dealing with an inoperable neoplasm. The biopsy however showed chronic inflammation, presumably tuberculous. The autopsy a few weeks later revealed a fixed mediastinum and large tracheobronchial nodes. The pericardium was 2 cm. thick and numerous areas of caseation necrosis were evident. Tubercle bacilli were isolated. Late in the disease, the ECG showed the changes of pericarditis.

LESIONS SECONDARY TO DISSEMINATED LUPUS ERYTHEMATOSUS

In a series such as this, and in a hospital with a 98 percent male patient population, it seems unusual that pericarditis would be due to disseminated lupus erythematosus in 4 patients. One patient was a 27-year-old woman; the others were men, aged 25, 47, and 58 years. Two died and the younger 2 were patients at the time of writing. All showed the typical diffuse involvement of tissues and organ systems, including the skin. The 2 who died had no remissions. The others have had numerous remissions and exacerbations. All had characteristic electrocardiographic changes. Three had friction rubs. Two had rather massive serosanguineous effusions which required tapping to relieve tamponade. The patient who was examined post mortem showed a fibrous adhesive pericarditis without fluid. The remaining patients had no evidence of pericarditis or endocarditis at the time of writing.

MISCELLANEOUS GROUP

The patient with a pericardial celomic cyst was 32 years old and had an asymptomatic mass in his chest discovered in a routine roentgenogram of the chest. The mass was round and homogeneous and thought to be a benign tumor. At operation the cyst was discovered and resected. The patient was asymptomatic 1 year after operation.

The patient with pericarditis secondary to subdiaphragmatic abscess was a 40-year-old man with peritonitis following perforation of a duodenal ulcer. Subdiaphragmatic abscess and bilateral pleural effusion developed. He complained of precordial pain and dyspnea and had a sinus tachycardia. No friction rub was heard but the ECG was typical and showed the usual evolution of pericarditis. He recovered completely.

The patient with hemopericardium and tamponade secondary to surgical trauma was a 34-year-old man who underwent gastric resection for a stenosing duodenal ulcer and following operation suddenly developed shock, dyspnea, friction rub, and tamponade. Pericardial taps on 2 occasions yielded several hundred cubic centimeters of blood. The ECG showed low QRS- and T-wave voltage. He recovered and was asymptomatic 2 years later.

The patient with polyserositis was a 62-year-old man who had been hospitalized on numerous occasions for congestive heart failure. On each occasion the roentgenogram showed cardiac enlargement and bilateral pleural effusion. Autopsy showed obliteration of the pericardial cavity. The membrane was 7 mm. thick and densely fibrous. The same fibrous change was found in the pleura and to a lesser extent in the peritoneum. The patient also had coronary artery disease and portal cirrhosis.

The patient with hemopericardium secondary to rupture of the aorta was a 59-year-old man who died suddenly and at autopsy showed idiopathic medial necrosis of the aorta with perforation of the proximal ascending portion. Six hundred and fifty cubic centimeters of blood were recovered from the pericardial sac.

The patient with pericarditis due to irradiation therapy had a mediastinal lymphoma which had been treated with large doses of x-ray directed at enlarged mediastinal lymph nodes. Terminally he complained of dyspnea and developed tachycardia. Autopsy showed thickening of the epicardium with fibrosis and hyperplasia of the epicardial cells.

PERICARDITIS OF UNDETERMINED ORIGIN

Only 2 of the 17 patients, who had pericarditis which could not be included in any of the foregoing categories, were incidentally discovered at postmortem examination and both had fibrous adhesions between the visceral and parietal pericardium. One died of bronchogenic carcinoma and the other of peritonitis. There was no evidence of rheumatic infection.

Four patients had acute pericarditis at the time of death. One was a chronic arthritic who had been bedfast for years and after an ingravescent course of many months, he rapidly developed cardiac tamponade. A roentgenogram showed a large cardiac silhouette. Several hundred cubic centimeters of thick pus were aspirated from the pericardial cavi-

ty. The culture showed *Staphylococcus aureus.* Two died of causes unrelated to the pericarditis; an early acute fibrinous pericarditis was found in 1 and purulent pericarditis in the other. The latter had both bronchogenic carcinoma and active tuberculosis but the pericarditis was nontuberculous and probably a terminal event. The fourth patient was a 36-year-old man who complained of feeling ill 5 days prior to admission and developed severe chest and epigastric pain. On admission he had a persistent tachycardia and a high sustained fever. He died a few days later. A shaggy fibrinous pericarditis with thickening of the pericardium and 200 cc. of amber pericardial fluid were found. The myocardium showed only minimal fibrosis. No other significant lesion was present in any organ and a diagnosis of rheumatic fever was not established.

Eleven patients, in whom no underlying disease or cause could be discovered, presented an almost identical clinical picture. All recovered. They complained of anterior chest pain and dyspnea beginning insidiously and becoming severe and continuous after a few days. Cough and diaphoresis were also present. All were acutely ill but had only a low-grade fever. Only 2 had a leukocytosis. Seven had elevated sedimentation rates. Four had friction rubs. The ECG's showed the typical changes and evolution of pericarditis in 9. Of these 9, all recovered within from 3 to 9 (average 6) weeks. The ECG's became normal and recovery was complete. Antibiotic and salicylate therapy did not seem to alter the course of the disease. The typical evolution of the ECG was the most important single diagnostic aid. The 2 patients who did not have electrocardiographic changes had the same clinical findings and course as the others and were seen during the same period. Seven of these patients had a history of upper respiratory infection 2 or 3 weeks before the onset of the chest pain. Two of the patients had congestive heart failure that responded to digitalization, oxygen, and diuretics. These patients were the only ones of this group to have enlarged hearts. The heart size became normal within 4 weeks. Pneumonitis was not present in any of these patients. The findings in this group of 11 patients corresponds closely to those of 27 patients reported by Levy and Patterson *(10).* Their patients recovered without residual symptoms. The follow-up period after recovery in our patients ranged from 2 months to 4 years during which time there were no recurrences or sequelas.

CONCLUSIONS

There seems to be no reliable clinical means of detecting neoplastic involvement of the pericardium prior to exploration.

The ECG was important in establishing the diagnosis in those patients in whom no cause for the pericarditis could be determined. The

(10) Levy, R. L., and Patterson, M. C.: Acute serofibrinous pericarditis of undetermined cause; study of 27 cases. Am. J. Med. 8: 34-45, Jan. 1950.

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course of the disease in these patients did not appear to be altered by antibiotic therapy.

Of 43 patients whose pericarditis was secondary to rheumatic fever, uremia, tuberculosis, pneumonia, and myocardial infarction, the diagnosis was made clinically in only 14 (5 rheumatic, 5 uremic, 2 pneumonic, and 2 tuberculous patients) because the lesion was either not considered or clinical symptoms and signs were absent.

Of 4 patients whose pericarditis was due to disseminated lupus erythematosus 2 died during the early stages. All developed effusion and 2 required tapping to relieve tamponade. All had electrocardiographic changes and 3 had friction rubs.

Of the entire group of 100 patients, the diagnosis was established clinically in only 29. The ECG was more valuable than physical examination or the roentgenogram in arriving at an ante-mortem diagnosis.

BOOK REVIEW

Brain Tumors of Childhood, by *Henry M. Cuneo,* M. D., Assistant Clinical Professor of Neurological Surgery, University of Southern California School of Medicine, Los Angeles, Calif., and *Carl W. Rand,* M. D., Clinical Professor of Neurological Surgery, University of Southern California School of Medicine, Los Angeles, Calif. American Lecture Series Publication No. 104, A Monograph in American Lectures In Surgery. 224 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $5.75.

This book brings the reader up to date on valuable recent additions to the literature and gives the conclusions to be derived from the authors' study of 83 children with brain tumor. It gives a short succinct enumeration of symptoms and a guide to differential diagnosis. The authors point out that the voluntary tilting of the head to one side may not indicate the side of a cerebellar astrocytoma but rather that it may be an effort of the child to prevent diplopia because of a paralyzed trochlear nerve; vertebral arteriographic study may show papillomas of the choroid plexus to be filled with the contrast media. They consider eosinophilic granulomas of the skull to be one form of reticuloendotheliosis (or xanthomatosis). They briefly discuss the diagnosis of infectious granulomas. Surgical technics are described only briefly. This pocket-sized book is recommended to the pediatrician, the neurologist, and the neurosurgeon.—*Lt. Comdr. G. Clark, MC, U. S. N.*

Ossified Dural Hematoma[(1)]

James W. Erchul, *Captain, MC, U. S. A.*

Harvey S. Rosenberg, *First Lieutenant, MC, U. S. A.*

A RARE end result in the natural course of chronic subdural hematoma is ossification. The physiologic effect of estrogen on bone is hyperossification *(2)*. The concomitant occurrence of an ossified dural hematoma in a patient after prolonged estrogen therapy suggests a possible intensification of the bone formation by the estrogen.

CASE REPORT

A 78-year-old man was hospitalized on 5 separate occasions over a period of 4 years for symptoms referable to the nervous and genitourinary systems. He was first seen because of an episode of syncope followed by a transient paresis of the right upper extremity. No history of trauma was elicited. Following his first admission he had periodic epileptiform seizures associated with mental disorientation. Because of his senility he was rarely mentally clear so that accuracy of clinical data was always questionable. Contributions to the history by members of his family were impeded by their inability to speak English. His personal habits included the consumption of up to a fifth of a gallon of whisky daily.

Neurologic examinations revealed fair cerebellar function. The Romberg test was slightly positive. There was some past pointing and marked intention tremor. There was generally poor muscular coordination. An EEG was read as suggestive of diffuse advanced cerebral arteriosclerosis with a secondary interpretation of psychomotor epilepsy and/or grand mal epilepsy and of atrophy of the cortex of the left temporal lobe.

He was given 3 capsules of dilantin daily with fair control. His initial symptoms (nocturia and urinary frequency) gradually progressed to a complete bladder neck obstruction, which required a suprapubic

(1) From the William Beaumont Army Hospital, Fort Bliss, Tex.

(2) Schilling, A.; Laszlo, P.; Bellin, J.; and Gottesman, E. P.: Effect of diethylstilbestrol on calcium, phosphorus, and nitrogen metabolism of prostatic carcinoma. J. Clin. Investigation 29: 918-924, July 1950.

prostatectomy for relief. The surgical specimen consisted of a moderately well differentiated prostatic adenocarcinoma. He was given from 6 to 10 mg. of diethylstilbestrol daily for the final 16 months of his life. There was clinical evidence of recurrence of the carcinoma 10 months after the prostatectomy and he died 16 months postoperatively with evidence of pyelonephritis and uremia.

Repeated complete blood counts, serologic tests for syphilis, and roentgenograms of the chest and skeletal system were all within normal limits. There was persistent gross and microscopic pyuria and hematuria. The nonprotein nitrogen was normal until the final admission when it rose to 205 mg. per 100 cc. The alkaline phosphatase was 6 Bodansky units. The acid phosphatase was 0.3 Shinowara-Jones-Reinhart units.

At autopsy the heart weighed 500 grams and exceeded 50 percent of the transthoracic diameter. Mild coronary sclerosis was observed but the valves were normal. A 5 by 3 cm. red-brown nonocclusive thrombus was located in the lumbar portion of the aorta. Moderate arteriosclerosis of this vessel and many of the major arteries was seen. The lungs together weighed 900 grams and were congested in dependent regions. The right main bronchus contained a small quantity of foreign material. Focal serosal hemorrhages were scattered along the small bowel serosa. The capsules stripped with some difficulty from the kidneys, which weighed 170 grams, exposing coarsely granular surfaces with occasional pits and scars. The remarkably pale sectioned surfaces displayed diminution of cortex. A cortical cyst, 1.5 cm. in diameter, occupied the upper pole of the left kidney. The congested, coarsely trabeculated mucosa of the urinary bladder was coated with purulent material. The remains of the prostate measured 4 cm. in diameter and were composed of exceedingly firm, pale tissue, which infiltrated the bladder neck but did not extend into other adjacent structures.

Two lead deposits, 0.7 and 0.5 cm. long, respectively, and 0.2 cm. thick, were imbedded superficially in the skull at the posterosuperior angle of the left squamous suture. No proliferation of the inner surface of the skull had occurred opposite this point. The dura over the left frontal lobe in a 10.5 by 6 cm. area, which began in a coronal plane just anterior to the lead, was thickened to an average of 0.35 cm. It stripped with the usual resistance from the skull and was not adherent to the arachnoid. Within this area were scattered 6 calcific foci from 0.5 to 1.0 cm. long and 0.3 cm. thick. The dura was brittle in these regions and leathery where calcium was absent. The inner dural surface was a speckled rust color. The brain was not flattened beneath the thickened dura and grossly showed only moderate sclerosis of the major vessels. No pressure conus, displacement of the ventricular system, or vascular lesions were found.

On microscopic section, moderate subintimal fibrosis was seen in the coronary arteries and focal fibrosis had occurred in the myocardium.

Foreign material occupied occasional bronchioles and alveoli, often surrounded by lipid-containing macrophages in the latter. Edema fluid and heart failure cells were abundant in some alveoli. Circumscribed focal areas of acute necrosis were scattered throughout the liver, marked by hepatic cell destruction and diffuse infiltration with neutrophils and lymphocytes. These foci were often central in position but could be seen in any portion of the lobule and were sharply demarcated from the surrounding normal parenchyma.

Pyelonephritis was manifested by stromal fibrosis, hyaline glomeruli, tubular casts, and chronic inflammatory cell infiltration in the parenchyma and beneath the pelvic mucosa. A moderate degree of arteriolar sclerosis was also present. The bladder neck and prostate were infiltrated by tumor elements represented by small tumor cells present singly, in small clusters, and in miniature acini. In some cell groups no cytoplasm was seen, but others had moderately abundant, clear cytoplasm. The absence or diminution of cytoplasm and the clustering of nuclei together with virtual absence of mitoses and cellular membranes was interpreted as an estrogenic effect. Marked atrophy of testicular tubular and interstitial elements had occurred. Aside from slight lipid depletion of the adrenal cortex, the endocrine glands were normal. No tumor cells were seen in the vertebras.

Although many large vascular spaces (figs. 1 and 2) were seen throughout the outer layer of the dura, these were mainly concentrated near the endosteal surface. Some of these measured several hundred microns in their greatest dimension and all lacked the elements of usual vascular channels, consisting rather of a single endothelial layer immediately adjacent to the surrounding dense hyalinized connective tissue. In the deeper layers was fully developed cancellous bone, often with marrow spaces containing fat and hematopoietic cells (fig. 1). A lesser number of vascular spaces lay adjacent to the inner surface of the ectopic bone in a less dense layer of connective tissue in which hemosiderin deposits, usually within phagocytes, were abundant and were located just beneath the inner surface of the dura (fig. 3).

Uremia secondary to pyelonephritis was the primary cause of death.

COMMENT

Although a clinical history of trauma was not obtained, it is believed that the presence of a foreign body, presumably a bullet, in the outer table of the skull at the left squamous suture is sufficient evidence that trauma had occurred. It could not be established, however, that this trauma was responsible for the ossified dural hematoma found at autopsy, or that this lesion was the cause of the convulsions. The possible predisposing effects of alcoholism on subdural hematoma has been discussed at length (3). The principal mechanisms postulated are de-

(3) Groff, R. A., and Grant, F. C.: Chronic subdural hematoma; collective review. Internat. Abstr. Surg. 74: 9-20, 1942; in Surg., Gynec. & Obst. Jan. 1942.

Figure 1. Trabecular bone with marrow spaces. Note vascular spaces in the hyalinized dura. The endosteal surface is above. Figure 2. Vascular spaces under greater magnification. Note proximity of the endothelium to the hyalinized dura. Compare the size of the lumen with the erythrocytes in upper center.

generative vascular changes, cystic degeneration of the dura (4), increased tendency to injury (5), and cerebral edema with increased susceptibility to laceration of the bridging veins (6).

The presence of vascular channels in the dura is frequently mentioned in pathologic descriptions. According to some (7), giant capillaries indicate a spontaneous origin and large vascular spaces similar to

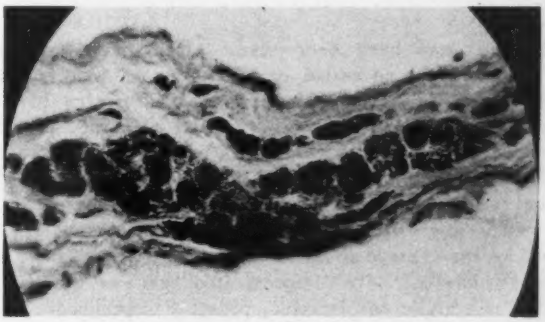

Figure 3. Hemosiderin-filled macrophages in dura adjacent to subdural space above. Space below is artefactual splitting.

those seen in this case would imply a traumatic cause. This means of differentiation is not universally accepted (8). The failure to demonstrate tumor cells in the outer capillary bed of the dura precludes a neoplastic origin as proposed by Russell and Cairns (9).

Case reports of calcified and ossified subdural hematomas have appeared sporadically in their literature and have been reviewed by Boyd and Merrell (10) Besides those reviewed in their report, several additional cases have appeared, chiefly in the foreign literature (11-13). Al-

(4) Wells, A. H., and Dickson, F. H.: Diagnostic case report (fibrolytic degeneration of meningeal dura with chronic intradural hematoma). Minnesota Med. 29: 253-258, Mar. 1946.

(5) Munro, D.: Cerebral subdural hematomas; study of 310 verified cases. New England J. Med. 227: 87-95, July 16, 1942.

(6) Leary, T.: Subdural hemorrhages. J. A. M. A. 103: 897-903, Sept. 22, 1934.

(7) Putnam, T. J., and Cushing, H.: Chronic subdural hematoma; its pathology, its relation to pachymeningitis hemorrhagica and its surgical treatment. Arch. Surg. 11: 329-393, Sept. 1925.

(8) Baker, A. B.: Subdural hematoma. Arch. Path. 26: 535-559, Aug. 1938.

(9) Russell, D. S., and Cairns, H.: Subdural false membrane or haematoma (pachymeningitis interna haemorrhagica) in carcinomatosis and sarcomatosis of dura mater. Brain. 57: 32-48, Mar. 1934. Quoted by Groff and Grant (3).

(10) Boyd, D. A., Jr., and Merrell, P.: Calcified subdural hematoma. J. Nerv. & Ment. Dis. 98: 609-617, Dec. 1943.

(11) Long, E.: Ossified subdural hematoma of traumatic origin. Zentralbl. f. Neurochir. 7: 193-202, 1943.

(12) Wertheimer, P., and Dechaume, J.: Les hématomes sousduraux calcifies. Acta psychiat. et neurol. 24: 731-742, 1949.

though no attempt is made to compute the general incidence of this phenomenon, it is of some significance that in 45 cases of solid type of chronic subdural hematoma reported by Munro *(5)*, only 1 was calcified and ossification was not mentioned. The incidence has been variously reported as "occasional," *(8)* "rare," *(10)* and "not unusual" *(14)*. Diagnosis based on roentgenologic technics could conceivably obscure the incidence because of the difficulty in distinguishing between bone and calcium *(15)*.

Subdural hematomas have been studied from many aspects without universal agreement as to cause or pathogenesis. Briefly, the controversy resolves itself into the questions of the relative incidence and significance of trauma and the site of initial bleeding. The concensus favors a trauma with subdural bleeding rather than a spontaneous origin and intradural bleeding as a cause. We have not attempted to clarify this issue but have illustrated one mechanism of repair. The lesion in this patient was compatible with either the chronic solid subdural hematoma of Munro *(5)* or the chronic dural hematoma of Baker *(8)*, which at this stage appear identical. The demonstrated process appears to be a manifestation of ectopic ossification, possibly superimposed on a focus of dystrophic calcification *(16)*. Many types of connective tissue not a part of the skeletal system possess latent osteogenic properties which may become manifest under certain conditions *(17)*. It is conceivable, therefore, that the fibroblastic tissues of the dura mater, having a common mesodermal origin with the skull *(18)*, should occasionally exercise osteogenic properties. The presence of normal acid and alkaline phosphatase values are of little significance because, according to Gomori *(19)*, acid phosphatase plays no role in calcification and calcification of hyaline connective tissue occurs without alkaline phosphatase activity.

Whatever the stimulus to formation of ectopic ossification or calcification, there is experimental evidence that administration of estrogens causes a "hyperossification" or increase in density of trabeculations in the subepiphyseal region of certain long bones. It has further been shown that this change induced by estrogen occurs independently of the parathyroid glands, although the presence of the pituitary is essential *(20)*.

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(13) Costa, A.: Sulla rare neoformazione di una calotta ossea, con mielopoiesi, nello spazio sottodurale. Arch "de Vecchi" per anat. pat. e med. clin. 2: 758-774, 1940.

(14) Camp, J. D.: Pathologic non-neoplastic intracranial calcification. J. A. M. A. 137: 1023-1031, July 17, 1948.

(15) Schüller, A.: Haematoma durae matris ossificans. Fortschr. a. d. Geb. d. Röntgenstrahlen 51: 119-124, Feb. 1935.

(16) Moore, R. A.: Textbook of Pathology. W. B. Saunders Co., Philadelphia, Pa., 1944. p. 71.

(17) Maximow, A. A., and Bloom, W.: Textbook of Histology, 5th edition. W. B. Saunders Co., Philadelphia, Pa., 1948. pp. 147-149.

(18) Leary, T., and Edwards, E. A.: Subdural space and its linings. Arch. Neurol. & Psychiat. 29: 691-701, Apr. 1933.

(19) Gomori, A.: Calcification and phosphatase. Am. J. Path. 19: 197-209, Mar. 1943.

(20) Baker, B. L., and Leek, J. H.: Relationship of parathyroid glands to action of estrogen on bone. Am. J. Physiol. 147: 522-526, Nov. 1946.

In an extensive metabolic study of a patient with prostatic carcinoma, Schilling et al. *(2)* demonstrated the calcium-retaining effect of estrogen as exemplified by a decrease in urinary and fecal excretion of calcium. Although the role of estrogen therapy in this case as related to the ossified dural hematoma cannot be dogmatically stated, its possible effect in intensifying the ossification cannot be ignored.

BOOK REVIEWS

Hippocrates on Intercourse and Pregnancy, An English Translation of On Semen and On the Development of the Child, by *Tage U. H. Ellinger,* Sc. D., M. A., with an introduction by *Alan F. Guttmacher,* M. D. 128 pages. Henry Schuman, Inc., New York, N. Y., publisher, 1952.

This is the first English translation of a portion of the 70 titles of the book collection known as the "Hippocratic Corpus." Although these particular sections were probably written not by the great Hippocrates of the Cos Medical School but by a physician from the rival Cnidian School, they are of particular interest to the student of medical history, the embryologist, and the biologist. The pre-Socratic Greek philosophers, unlike their Egyptian predecessors, were much interested in embryologic phenomena and in this writing the pangenetic hypothesis of Darwin was stated clearly about 2,300 years before the "Origin of the Species." Many other observations on embryology, heredity, and allied subjects which are still valid were also made and, generally, the old saw "There is nothing new in medicine" has further confirmation.

—*Lt. Col. H. C. Boyd, MC, U. S. A.*

Lippincott's Handbook of Dental Practice, edited by Louis I. Grossman, D. D. S., D. M. D. Authors: *W. Harry Archer,* M. S., D. D. S., *Carlisle C. Bastian,* D. D. S., *Basil G. Bibby,* B. D. S., Ph. D., D. M. D., *Allan G. Brodie,* D. D. S., Ph. D., *Lester W. Burket,* D. D. S., M. D., *James R. Cameron,* D. D. S., *Edward C. Dobbs,* D. D. S., *Edward M. Gre vatt,* D. D. S., *Louis I. Grossman,* D. D. S., D. M. D., *A. O. Klaffenbach,* D. D. S., *Benjamin Kornfeld,* D. D. S., *Howard K. Mathews,* D. D. S., *Balint Orban,* M. D., D. D. S., *Edward C. Stafne,* D. D. S., *J. J. Stetzer, Jr.,* D. D. S., *Charles A. Sweet,* D. D. S., *V. R. Trapozzano,* D. D. S., *Joseph F. Volker,* D. D. S., Ph. D., *Gordon R. Winter,* D. D. S. 2d edition. 503 pages; 388 illustrations; 4 color plates. J. B. Lippincott Co., Philadelphia, Pa., publisher, 1952. Price $10.

The second edition of this excellent work is greatly improved by the addition of a chapter on oral diagnosis, color plates in the section on periodontics, and a slight revision of the section on exodontics to include postoperative treatment and consideration of cysts. Also, Kornfeld has added a very complete section on direct resinous restorations. For the benefit of those who may not be familiar with the first edition, this

book represents essential knowledge for all practitioners of dentistry, presented in a clear concise manner by outstanding authors in each specialty. The book is well edited and there is no better ready reference for all the essentials of dental practice.

—*Lt. Col. J. P. Kane, U. S. A. F. (DC)*

The Internship, by *Roscoe L. Pullen,* A. B., M. D., F. A. C. P., Professor of Graduate Medicine; Director of the Division of Graduate Medicine; Vice-Dean, Tulane University of Louisiana School of Medicine; Senior Visiting Physician, Charity Hospital of Louisiana at New Orleans; Consultant in Medicine, Veterans Administration Hospital, New Orleans; Consultant to the Surgeon General, Department of the Army, Washington, D. C. American Lecture Series Publication No. 154, A Monograph in American Lectures in Internal Medicine. 35 pages. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $1.25.

This booklet will provide a modern concept of the status of internships within the United States as relates to university teaching hospitals, other civilian institutions, and the government services hospitals. The author considers the important factors which influence the educational experience obtained by an acceptable approved internship. He discusses the effects of modern hospitalization, including the voluntary prepayment insurance programs and the current popular trend of specialization. He recommends a junior internship during the fourth year in medical school in order to relieve the shortage of interns in this country. This would also enrich the educational value of the fourth year of medical school but it would place the senior internship on a residency basis and lessen the number of current internship spaces. This proposal needs further study before being widely adopted. The author believes that the rotating type of internship offers the best educational experience.

All members of the Professional Education and Training Committee and the members of the various hospital education committees should read this booklet carefully because it will give them an insight into the operation of an approved internship. It is recommended for the chiefs of services and others responsible for the training of interns.

—*Col. F. L. Wergeland, MC, U. S. A.*

The Present Status of Antibiotic Therapy, With Particular Reference to Chloramphenicol, Aureomycin, and Terramycin, by *Francis G. Blake,* M. D., Department of Internal Medicine, Yale University School of Medicine, New Haven, Conn. Augustus B. Wadsworth Lecture, May 16, 1951. American Lecture Series Publication No. 137, A Monograph in American Lectures in Tests and Techniques. 26 pages. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $.90.

The practitioner who infrequently treats patients suffering from acute infectious disease will find this booklet helpful in bringing him up to date on the current status and the great value of chloramphenicol, aureomycin, and terramycin as therapeutic tools in our ever-increasing armamentarium against infectious agents.

—*Lt. Col. W. J. Walker, MC, U. S. A.*

Electrocardiographic Abnormalities in Wolff-Parkinson-White Syndrome[1]

Charles I. Hamilton, Jr., *Major, U. S. A. F. (MC)*
William N. St. John, *Major, U. S. A. F. (MC)*
Robert B. Milligan, *Captain, U. S. A. F. (MC)*

A REVIEW of the pertinent available literature revealed that the occurrence of RS-T segment and T-wave abnormalities in the Wolff-Parkinson-White syndrome are accepted as an expected variation. The purpose of this article is to discuss these factors as they applied to 2 patients with underlying heart disease. Bundle-branch block with a short P-R interval (Wolff-Parkinson-White syndrome) is not unique. The electrocardiographic pattern has been frequently described as one in which the P-R interval is abnormally short (0.1 seconds or less) and the QRS complex is wide *(2)*. The net result is an apparent bundle-branch block secondary to an aberrant conduction mechanism between the auricle and ventricle which activates one ventricle before the other, or produces an electrocardiographic pre-excitation wave followed by more or less synchronous ventricular stimulation.

This syndrome is not indicative of heart disease and was more often than not the sole abnormality in cases heretofore reported. Following are 2 cases associated with heart disease in which the character of the ECG was such that the RS-T segment and T-wave abnormalities could not necessarily be attributed to the bundle-branch block.

CASE REPORTS

Case 1. A 26-year-old airman entered this hospital for evaluation of a heart murmur. He reported that he had been hospitalized when he was 16 years old because of acute polyarthritis and fever. During this

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(1) U. S. Air Force Hospital, Lackland Air Force Base, San Antonio, Tex.

(2) Wolff, L.; Parkinson, J.; and White, P. D.: Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. Am. Heart J. 5: 685-704, Aug. 1930.

illness a heart murmur, not further described, was noted. The left heart border and point of maximum impulse were in the fifth left interspace, 6 cm. to the left of the left sternal border. The first mitral sound was markedly accentuated and preceded by a short, low-pitched presystolic murmur best heard with the patient in the left lateral position. There was a moderately high-pitched systolic aortic murmur and a soft, blowing apical systolic murmur. The blood pressure and pulse rate were normal.

A roentgenogram and fluoroscopic examination of the heart revealed increased prominence of the left atrium and ventricle. The transverse diameter of the heart was 13 percent above the predicted size for the patient's height and weight. The ECG (fig. 1) had a P-R interval of 0.1 seconds and a QRS complex measuring from 0.12 to 0.14 seconds with slurring in the upstroke of the R wave in all precordial leads. There was a moderate counterclockwise rotation, high voltage of R waves in leads V_3 and V_4 (35 mm.) with depression of the RS-T segment and asymmetrically inverted T waves. The ECG fulfills the criteria set for the Wolff-Parkinson-White syndrome. In addition, it was decided that the high voltage R waves, depression of the RS-T segment, and asymmetrical T-wave inversion met the standards for left ventricular hypertrophy enumerated by Sokolow and Lyon (3). There was suggestive confirmatory evidence by cardiofluoroscopy which revealed left ventricular enlargement.

The patient was discharged with a diagnosis of rheumatic heart disease with (1) mitral and aortic stenosis and mitral insufficiency; (2) cardiomegaly manifested by enlargement of the left ventricle and auricle; (3) sinus rhythm, compensated; and (4) class 1 cardiac function. The additional diagnosis of Wolff-Parkinson-White syndrome was not known to be related to the rheumatic heart disease.

Case 2. A 38-year-old airman complained of moderate exertional dyspnea and recurrent joint pain of 3 year's duration. In 1948 he had been hospitalized for 6 weeks because of painful, swollen, red, and hot joints (the left hip, knee, and ankle). No hospital records for this period were available and the patient did not know what diagnosis, if any, had been made. Since that time he had experienced intermittent pain in the left hip, knee, and ankle which was usually exacerbated by cold or damp weather. In 1950 he was hospitalized for these complaints and subsequently told that he had an enlarged heart due to rheumatic heart disease. Though other historical factors of potential interest were sought for, the remainder of his history was negative.

His blood pressure was 130/90 in both arms. The pulse rate was 110. The area of cardiac dullness extended to the anterior axillary line in the fifth left interspace. Retrosternal cardiac dullness was also elicited.

(3) Sokolow, M., and Lyon, T. P.: Ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. Am. Heart J. 37: 161-186, Feb. 1949.

A roentgenogram revealed an unusually wide and tortuous aorta. The transverse diameter of the heart was more than 20 percent above the predicted diameter for his height and weight. Cardiofluoroscopy confirmed the above findings and established that enlargement of

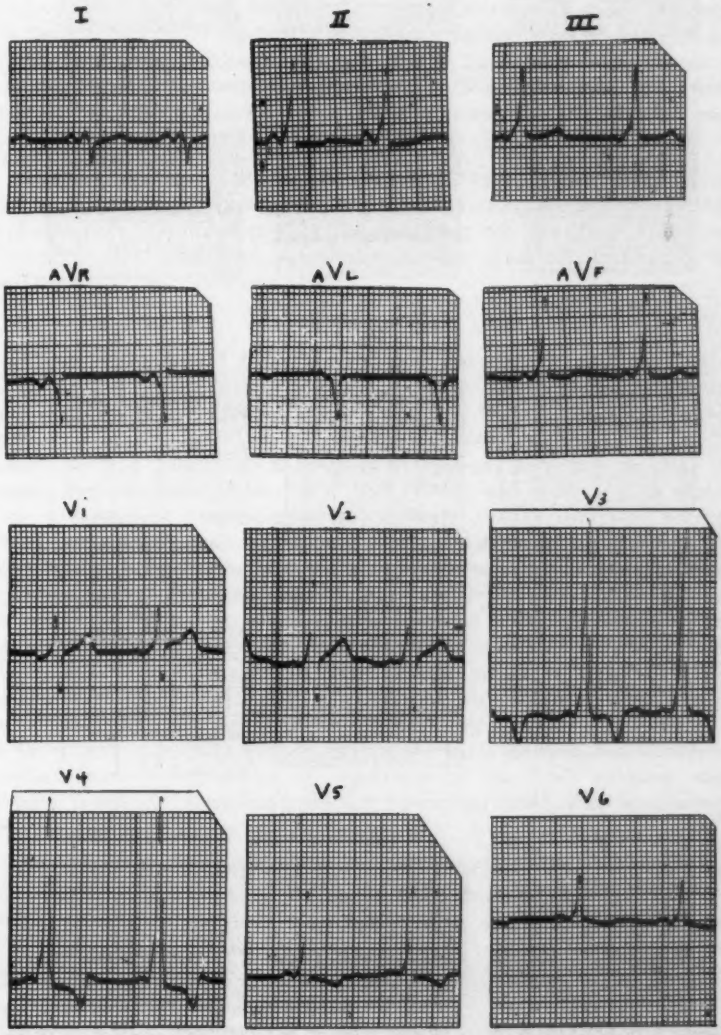

Figure 1. In addition to the short P-R interval and wide QRS complex, note abnormalities in leads V3 to V6 consistent with left ventricular hypertrophy.

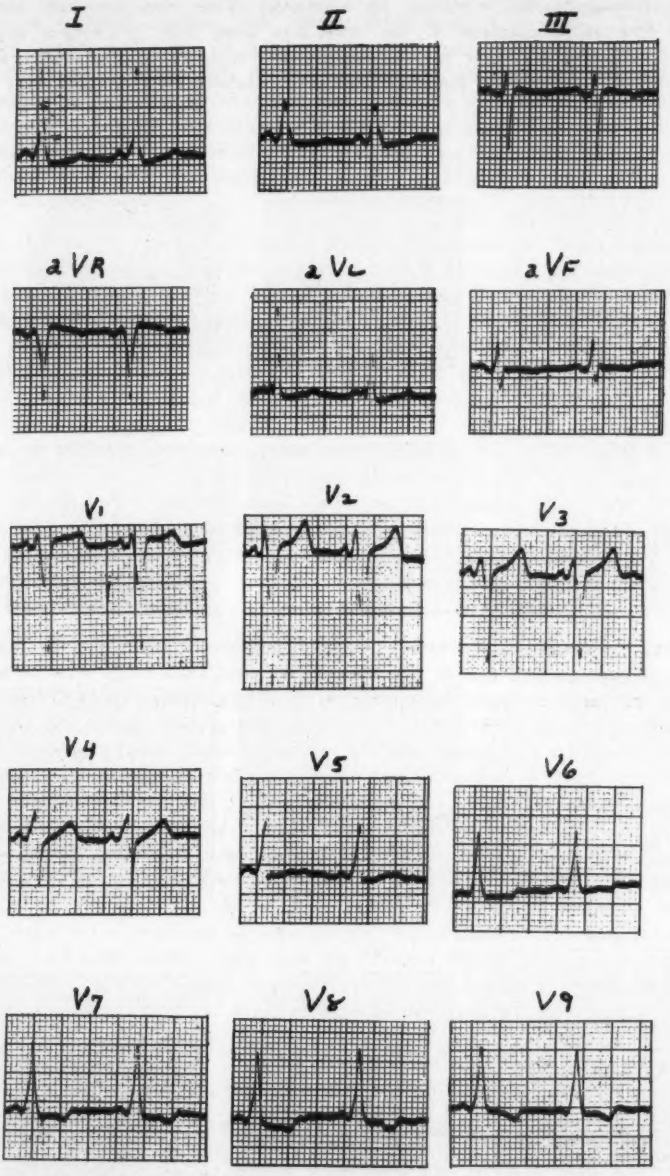

Figure 2. In addition to the short P-R interval and wide QRS complex, note the high R wave in lead aV$_L$ with RS-T segment and T-wave abnormalities consistent with left ventricular hypertrophy.

the left ventricle was the principal manifestation of the cardiomegaly. The ECG (fig. 2) revealed a P-R interval of 0.1 seconds and a QRS complex measuring 0.12 seconds in lead 2. The QRS complex in the precordial leads measured 0.14 seconds. These findings met the established criteria for the Wolff-Parkinson-White syndrome. Additional findings of significance were depression of the RS-T segment with convexity upward and diphasic to asymmetrically inverted T waves in the left precordial leads with significant height of the R wave, i. e., R in lead aV_L exceeded 13 mm. These findings would support a diagnosis of left ventricular hypertrophy. The substantial left ventricular enlargement demonstrated cardiofluoroscopically offered reasonable support for such a diagnosis.

The patient was returned to duty with a diagnosis of possible rheumatic heart disease, manifested by (1) cardiomegaly (left ventricular enlargement); (2) sinus rhythm, compensated; and (3) class 1 cardiac function. The Wolff-Parkinson-White pattern was not known to be related to the heart disease.

DISCUSSION

Abnormalities of the RS-T segment and T wave are not a necessary component of the electrocardiographic pattern of short P-R intervals and widened QRS complexes. Probably half of these will reveal T-wave inversion at least. Willius and Carryer *(4)* found variations in the T waves in 46.1 percent of a group of 65 patients with this pattern. In these, the reciprocal direction of the T wave was sufficient to simulate a pattern of bundle-branch block. When T-wave inversion occurs it is probably a result of alteration in repolarization as effected by asynchrony, i. e., contraction of one ventricle at an appreciable interval after the other. This has been demonstrated by Tamagna et al. *(5)* in fusion experiments by variable pre-excitation of the right ventricle of the normal cat heart. Of further interest was the revelation that the shorter the resultant P-R interval, the greater was the T-wave inversion. This experiment was performed with the limb leads only in which significant deviation of the RS-T segment was not revealed. It is entirely possible that a similar experiment carried out with precordial leads would result in depression of the RS-T segment and T-wave inversion in, at least, the left precordial leads, thus simulating left bundle-branch block.

Depending on the nature of the aberrant auricular-ventricular pathways, asynchrony may exist in some persons with the Wolff-Parkinson-White pattern. Asynchrony is not a necessary component and may, in

(4) Willius, F. A., and Carryer, H. M.: Cardiac clinics; electrocardiograms displaying short P-R intervals with prolonged QRS complexes; analysis of 65 cases. Proc. Staff Meet., Mayo Clin. 21: 438-444, Nov. 13, 1946.

(5) Tamagna, I. G.; Butterworth, J. S.; and Poindexter, C. A.: Wolff-Parkinson-White syndrome with myocardial infarction: experimental study. Am. Heart J. 35: 948-952, June 1948.

fact, be absent (6). There could, conceivably, be other factors accounting for the RS-T segment and T-wave variations which may be revealed by further investigation. It would, thus, seem important not to regard these variations as an expected variant of anomalous auriculoventricular pathways. Demonstration of asynchrony, as with the electrokymogram (6), probably permits the assumption that the RS-T segment and T-wave changes are not a manifestation of heart disease. Barring revelations of other normal physiologic explanations, the absence of asynchrony, necessitates diligent search for evidence of heart disease.

CONCLUSION

Two patients are presented who, in addition to having a Wolff-Parkinson-White syndrome, had definite evidence of heart disease associated with RS-T segment and T-wave abnormalities in the ECG. It is suggested that (1) such variations are not a necessary component of the pattern characterized by short P-R interval and wide QRS complex, (2) in the absence of asynchrony they may be a manifestation of heart disease, and (3) their presence necessitates excluding heart disease.

(6) Dack, S.; Paley, D. H.; and Brahms, S. S.: Electrokymogram in Wolff-Parkinson-White syndrome; study of left and right ventricular ejection. Am. Heart J. 41: 437-447, Mar. 1951.

BOOK REVIEW

The Photography of Patients, Including Discussions of Basic Photographic and Optical Principles, by *H. Lou Gibson,* F. B. P. A., A. P. S. A., Medical Division, Eastman Kodak Company, Rochester, N. Y. American Lecture Series Publication No. 95, A Monograph in American Lectures in Medical Photography. 118 pages; 93 illustrations (4 in color). Charles C Thomas, Publisher, Springfield, Ill., 1952.

This small, well-written volume serves a dual purpose. It offers the physician a systematic approach to the photography of patients and the medical photographer with a much needed reference. The author has graphically put first things first. The information contained herein is practical and devoid of superfluity. By the skillful use of photographs he has been able to explain a technical subject with a minimum of words. This alliance of words and photography is best illustrated in the chapters on lens properties, positioning, and lighting. These chapters offer a sound foundation for the pursuance of other branches of medical photography. The book is well printed and the reproductions are of the best quality.—*F. W. Miller*

Sporotrichosis[1]

Paul M. Walstad, *Captain, MC, U. S. A. R.*

Stephen M. Gelenger, *Lieutenant Colonel, MC, U. S. A. R.*

THE mycotic infection, sporotrichosis, which rarely affects man is caused by the fungus, *Sporotrichum schenckii.* The initial infection is transmitted by plants or animals, both of which are commonly infected with the fungus. Infection in man is usually through a break in the skin, such as a puncture wound of a thorn, which is frequently disregarded or unnoticed until a painful localized area of tenderness and redness appears. As the disease develops, gummalike, painless nodules, in chains extending proximally, appear along the lymphatic pathways of the extremity. These necrotize centrally, producing craterlike lesions which discharge purulent material. The peripheral borders of the lesions are circumscribed and indurated. Less frequently seen are the disseminated cutaneous and extracutaneous form of sporotrichosis (2).

Clinically it may be difficult to distinguish sporotrichosis from syphilis, tuberculosis, tularemia, or infection with a gram-positive organism such as staphylococcus. The diagnosis is established by growing cultures from the discharge on Sabouraud's medium at room temperature. Biopsy specimens or smears of the discharge will not reveal the organism. Microscopically the fungus appears as a branching hyaline structure with short lateral branches containing groups of spores distally (3).

The disease has been reported from 29 states, but appears to be more prevalent in the states west of the Mississippi. The first reported case of sporotrichosis from New Hampshire occurred in 1950 (4).

To allow the disease to go undiagnosed is to leave the patient with a prolonged disabling condition. An incorrect diagnosis of tuberculosis or syphilis is equally bad, causing the patient unjustified anxiety. With the correct diagnosis established, treatment can begin at once with

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(1) Specialized Medical Treatment Center, U. S. Army Hospital, Camp Cooke, Calif.

(2) Gastineau, F. M.; Spolyar, L. W.; and Haynes, E.: Sporotrichosis; report of 6 cases among florists. J. A. M. A. 117: 1074-1077, Sept. 27, 1941.

(3) Boyd, W.: Surgical Pathology. 5th edition. W. B. Saunders Co., Philadelphia, Pa., 1942. pp. 62-63.

(4) Cole, D. P., and Lobitz, W. C., Jr.: Sporotrichosis in New Hampshire. New England J. Med. 243: 132-133, July 27, 1950.

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potassium iodide which is remarkably effective in bringing about recovery. As yet, no better form of therapy has been discovered (5).

CASE REPORT

A 28-year-old soldier reported to the dispensary on 15 January 1951 because of a tender, swollen, right fourth finger. Several days earlier he had noted a small, tender, localized area on this finger which had apparently developed without relation to any known injury. He was given penicillin intramuscularly for 3 days. Warm saline compresses were applied to the hand. Improvement was only temporary as 2 weeks later the finger again became red and edematous. A fluctuant area was incised and drained of purulent material, following which improvement again occurred. Then 1 month after the soldier's first appearance at the clinic, indurated nodules of the right forearm and arm became apparent.

When admitted to the hospital on 12 March, because of continued tenderness and swelling of the right fourth digit, the patient had a necrotic ulcer with purulent drainage of the extensor surface of the right fourth digit. There were about 25 circumscribed elevated nontender lesions arranged in linear fashion along the dorsal lateral surface of the right forearm and arm. No axillary lymph nodes were palpable. The patient's temperature was 99.2° F. This man had been stationed in California during the preceding year. In November 1950 he had been deer hunting. Early in January 1951 he was on maneuvers in scattered, wooded areas. He believed that he might have pricked his finger with a pine needle at this time or that he might have punctured the skin with a thorn while working in his garden outside the Base, but no definite date of injury could be established.

The leukocyte count was 11,500 with 71 percent neutrophils. Agglutination tests for typhoid, paratyphoid, tularemia, and brucellosis were negative. Bacteriologic culture of the discharge revealed only gram-positive cocci resembling staphylococci. Mycologic cultures from the finger lesion and one of the nodules obtained by aspiration showed no growth on Sabouraud's agar at 37° C. The biopsy specimen from one of the nodules showed only nonspecific inflammatory, necrotic tissue.

During the following 3 weeks all nodules became necrotic and suppurative. Alternate courses of penicillin and aureomycin were begun but had minimal effect on the widespread, openly discharging lesions (fig. 1). Because clinically the case resembled sporotrichosis so closely, repeated cultures from the depths of the lesions were made until a pure growth of *Sporotrichum schenckii* was obtained on Sabouraud's medium at room temperature. With the diagnosis firmly established, specific therapy for sporotrichosis was initiated. This consisted of increasing doses of a saturated solution of potassium iodide in cream given orally

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(5) Muskatblit, E., and Tulipan, L.: Sporotrichosis. Arch. Dermat. & Syph. 63: 385-386, Mar. 1951.

Figure 1. Lesions before specific treatment.

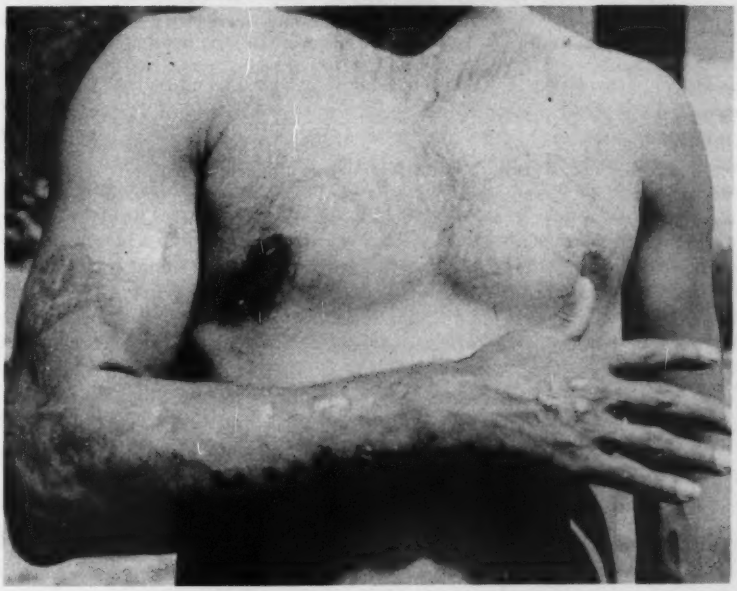

Figure 2. After 8 weeks' treatment. Scars mark sites of former suppurative lesions.

in divided doses until a dose of 35 drops t. i. d. was reached. Dressings saturated with Lugol's solution were applied daily to the lesions. Improvement was noted within 1 week and 8 weeks after specific therapy was started all lesions were completely healed (fig. 2). Six months later there was no evidence of recurrence.

SUMMARY

Sporotrichosis is a fungus infection which usually follows a minor trauma. Its onset is insidious but its course is prolonged and disabling if untreated. It must be considered in the differential diagnosis of chronic, suppurative lesions of the extremities. The diagnosis is established by growing cultures of the organism on Sabouraud's medium at room temperature. Biopsy or smear is of no value in detecting the fungus. The administration of potassium iodide brings about prompt healing.

BOOK REVIEW

Annual Reprint of the Reports of the Council on Pharmacy and Chemistry of the American Medical Association, With the Comments That Have Appeared in the Journal of the American Medical Association 1950. 106 pages. J. B. Lippincott Co., Philadelphia, Pa., publishers, Price $2.

The reports of the Council on Pharmacy and Chemistry of the American Medical Association are so well known to members of the medical profession that they need no introduction. This volume is made up of 18 reports authorized for publication in 1950, some of which have not previously been published. Reports on the use of antihistaminic agents in prophylaxis and treatment of the common cold, the status of BCG vaccine, the status of chemotherapy of tuberculosis, the pharmacology of certain insecticides, and the therapy of mammary cancer metastasis to bone are included. By collecting these reports into a single volume annually, the Council has made available in convenient form current reviews of important therapeutic agents. This is a useful reference for all practitioners of medicine.—*Maj. J. L. Sherman, Jr., MC, U. S. A.*

Malignant Mediastinal Teratoma

Joseph M. Hanner, *Commander, MC, U. S. N. (1)*
Frank S. Ashbum, *M. D. (2)*
Raymond J. Leffler, *Lieutenant, junior grade, MC, U. S. N. (1)*

A MALIGNANT teratoma in the mediastinum is an uncommon tumor. During World War II the Armed Forces Institute of Pathology acquired 29 cases of teratoma located in the mediastinum but only about one-third of these were malignant *(3)*.

CASE REPORT

A 19-year-old marine was admitted to the hospital following a routine roentgenogram of the chest which revealed a tumor in the right side of the chest. He was ready for discharge from the Marine Corps and minimized his symptoms. It was after a great deal of questioning that it was found that he had dyspnea, cough, and some substernal pain. For 3 months he had been short of breath and for 3 weeks his face had been swollen in the mornings. In stooping forward he noted dizziness. A dry cough associated with a dull substernal pain had been present for about 1 month. These symptoms were aggravated by lying down. The pain was not severe but traversed his chest anteroposteriorly from the fourth interspace to the midscapular area, then radiated to the back of his neck. His appetite was good. There was no hemoptysis.

Dyspnea was evident when the patient was lying down. His neck was full and the veins of the neck were prominent bilaterally. There was no edema. The skin of his neck and the upper part of his chest was red when he assumed a reclining position. The right side of his chest was dull to percussion and the voice sounds were decreased on this side. Venous pressure was 54.8 in his left arm. His sedimentation rate was 14. Sputum on 3 occasions was negative for acid-fast bacilli. His vital capacity was 80 percent. A roentgenogram of the chest (fig. 1) revealed

(1) U. S. Naval Hospital, National Naval Medical Center, Bethesda, Md.
(2) Consultant in Thoracic Surgery, U. S. Naval Hospital, National Naval Medical Center, Bethesda, Md.
(3) Schlumberger, H. G.: Tumors of the Mediastinum. Armed Forces Institute of Pathology, Washington, D. C., 1951. Section 5, Fascicle 18. p. 49.

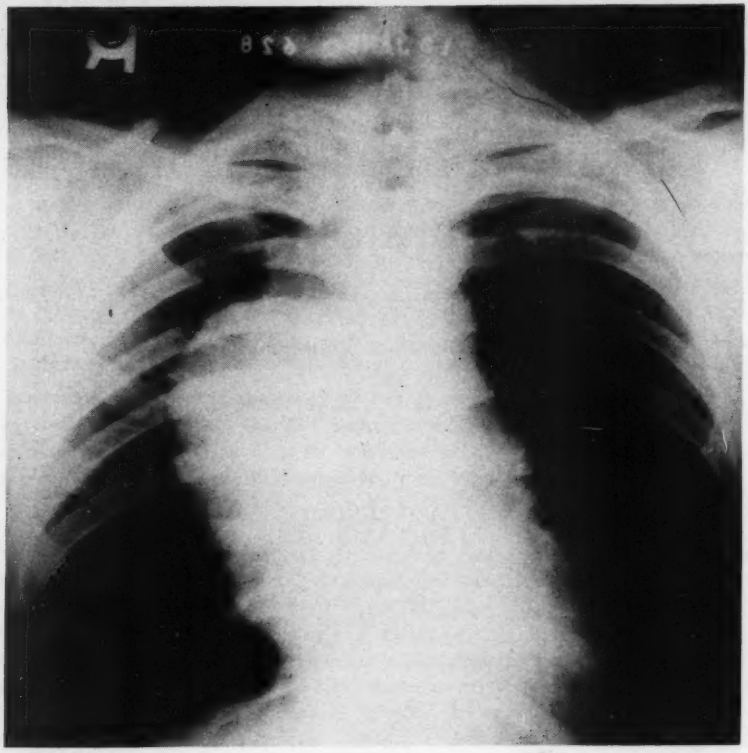

Figure 1. Roentgenogram showing mediastinal mass protruding into right side of the chest.

a mass in the anterior mediastinum protruding into the right side of the chest compressing the lung and causing an atelectasis in the upper and middle lobes of the right lung.

Fifteen days after admission an exploratory thoracotomy was performed on the right side. On opening the thoracic cavity about 500 cc. of bloody fluid was found. An inoperable tumor involving the anterior mediastinum was seen. The tumor protruded into the right lung and compressed the upper and middle lobes. It was adherent to the aorta and appeared to be invading the pericardium. Because needle aspiration freely yielded arterial blood the tumor was thought to be vascular in origin. It was deemed unwise to take a biopsy specimen and the chest was closed.

The patient recovered from the operation and was later given deep x-ray therapy with some relief of symptoms, but 1 month after the opera-

tion the dyspnea, cough, substernal pain, and dizziness returned and increased in severity. Edema with markedly dilated veins of the upper part of the thorax and face appeared. The patient died 3 months after admission and about 6 months after the onset of symptoms.

The autopsy revealed an oval mass originating in the anterior mediastinum. The mass was freed from the right chest wall. It measured 17 by 12 by 10 cm. The pericardial cavity was pushed downward and to the left. The tumor was surrounded by a fairly well-formed fibrous capsule but it had extended through the capsule into the pericardial cavity near the great vessels, into the superior vena cava, and posteriorly into the superior mediastinum (fig. 2). On section the tumor was honeycombed with cystic spaces, which contained a mucosanguineous material, and showed evidence of necrosis. The latter may have occurred as a result of the x-ray therapy (fig. 3). The tumor tissue was well vascularized. The line of junction with the lung and aorta was sharp and did not extend into the pulmonary tissue. The pericardial cavity was distended and

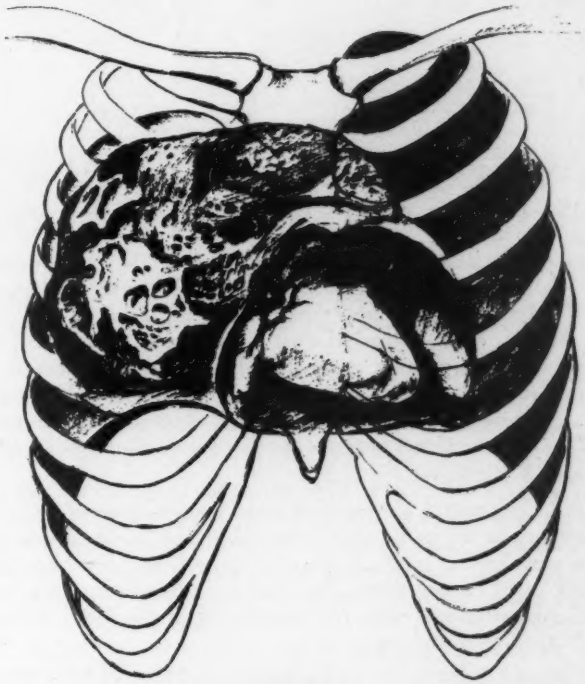

Figure 2. Drawing showing position of tumor in the chest.

measured 12 by 10 by 10 cm. It contained 600 cc. of fluid blood. The source of bleeding was from several vascular nubbins of the mass extending into the cavity about the great vessels (fig. 4).

Figure 3. Cross sections of tumor showing necrosis of tissue.

The heart was small and weighed 250 grams. The predominate microscopic finding was an angiomatous neoplasm with malignant cells lining the vascular spaces and occasionally occurring in the interstitial spaces (fig. 5). Many epithelial cystic structures were noted, some resembling respiratory tract with pseudostratified, columnar, ciliated epithelium (fig. 6). Other parts of the tumor contained a nonspecific cuboid, columnar, or flat epithelium. Cartilage was seen in several areas (fig. 7). Occasional pigment particles were seen, some resembling carbon and hemoglobin.

The tumor suggested angiosarcoma but was considered to be a malignant teratoma on the basis of its epithelial and cartilaginous elements. Invasion was limited to the pericardial cavity, the great vessels of the heart, and local lymph nodes. The immediate cause of death was from a cardiac tamponade resulting from the oozing of the blood from the pericardial extension of the tumor.

COMMENT

Because a tumor in the mediastinum lies in close relationship with such vital structures as the esophagus, vagus nerve, trachea, bronchi,

heart, great vessels, and sympathetic nerves, it may produce a wide array of symptoms involving several systems. Pressure on the esophagus will cause dysphagia and regurgitation of food. Involvement of the vagus may be noted by additional gastrointestinal symptoms such as vomiting. In some patients severe hiccups occur due to the phrenic nerve irritation. There are usually predominant respiratory manifestations such as dyspnea and cough. The tumor may exert traction on the trachea or bronchi in such a manner as to produce orthopnea. If the recurrent laryngeal nerve is affected by the tumor there will probably be stridor, brassy cough, and hoarseness.

Figure 4. Gross specimen showing extension into pericardial cavity at left. The lung is atelectatic and pushed posteriorly.

In addition to the gastrointestinal and respiratory symptoms, there are those resulting from the involvement of the vascular structures. Pressure and displacement of great vessels may cause cyanosis, edema, dilation of the veins of the neck and upper part of the thorax, dizziness, and headache. If the sympathetic nerves are affected there may be mitosis of the pupils with flushing and sweating of the face. There is usually substernal pain which may be referred to the back. In contrast to patients with malignant tumors elsewhere, death usually occurs within 1 year of their discovery and there is not time for emaciation to develop.

There are many theories regarding the origin of mediastinal teratomas. Schlumberger believed that they originate within the anlage of the thymus. Others believed that they spring from a segmented blastomere or

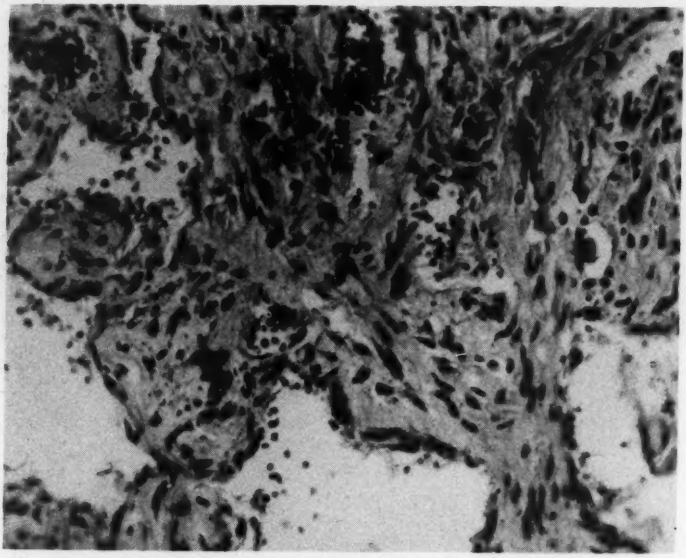

Figure 5. Section showing angiosarcomatous tissue with vascular spaces lined by malignant endothelial cells.

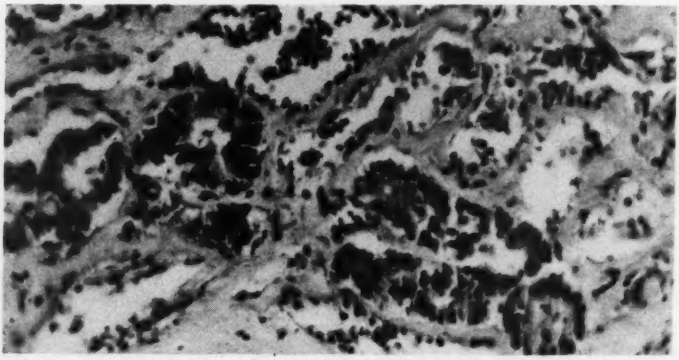

Figure 6. Section showing epithelial structures.

some malformation of the mediastinum (*4*). The tumor may be cystic or solid. It is usually noted in young men and occurs most frequently in the anterior mediastinum. Histologically the lining cells may be col-

(*4*) Boyd, W.: Surgical Pathology. 6th edition. W. B. Saunders Co., Philadelphia, Pa., 1947. p. 797.

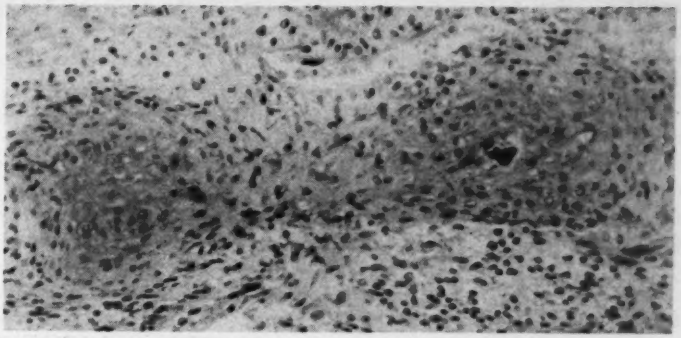

Figure 7. Section showing developing cartilage.

umnar or of the stratified squamous type. The contents of the tumor may be sebaceous, pilose, glandular, cartilaginous, osseous, or nervous tissue. Such a teratoid origin of tissue leads to an adenocarcinoma or epidermoid carcinoma. The malignancy is evidenced by invasion into nearby structures, such as the heart or great vessels, and this usually is the cause of death.

The case here reported demonstrates several of the characteristic symptoms and pathologic findings seen in malignant teratoma of the mediastinum.

BOOK REVIEWS

Signs and Symptoms, Applied Pathologic Physiology and Clinical Interpretation, edited by *Cyril Mitchell MacBryde,* A. B., M. D., F. A. C. P., Associate Professor of Clinical Medicine, Washington University School of Medicine; Assistant Physician, the Barnes Hospital; Director, Metabolism and Endocrine Clinics, Washington University Clinics, St. Louis, Mo. 2d edition. 783 pages; 98 illustrations; 50 charts; 8 color plates. J. B. Lippincott Co., Philadelphia, Pa., publisher, 1952. Price $10.

This new edition comes out 5 years after the first. The subtitle has been expanded and improved and the pages are smaller. In addition to revising and rearranging the chapters appearing in the first edition the following chapters have been added (1) Pathologic Bleeding by Benjamin Alexander, (2) Pigmentation of the Skin by Edward Edwards and Harold Jeghers, (3) Vertigo and Dizziness by H. H. Hyland, and (4) Clubbed Fingers and Hypertrophic Osteoarthropathy by Bernard Lipman and Edward Massie. These additions greatly enhance the value of the book. The chapter on edema has been rewritten by Henry

Schroeder. Many helpful diagrams and illustrations have been added. The editor has contributed several chapters as in the previous edition, including an excellent introductory chapter on history taking. In fact, the emphasis throughout the book is on careful history taking and basic knowledge of the underlying pathologic physiology. This book differs from French's classic "Index of Differential Diagnosis" in that the "Index," a much large book, gave a brief discussion of every conceivable sign or symptom and the present volume gives a very detailed discussion of about 40 of the most important signs and symptoms encountered in everyday practice. This book should prove invaluable to physicians engaged in almost any branch of medical practice. Each chapter is followed by either a bibliography or a list of references and most chapters contain a very useful summary or conclusion. An exception is the brief summary of the chapter on thoracic pain which says nothing which is not implicit in the title of the chapter and which should therefore have been omitted. The index is adequate.

—*Col. W. G. Brandstadt, MC, U. S. A.*

X-ray Interpretation, by *H. Cecil H. Bull,* M. A., M. B., M. R. C. P., Honorary Consulting Radiologist to the Royal Waterloo Hospital, London, and the General Hospital, Southend-on-Sea, with a Chapter on Radiography of the Head, by *James W. D. Bull,* M. A., M. B., M. R. C. P., D. M. R., Assistant Radiologist, St. George's Hospital; Radiologist, Maida Vale Hospital for Nervous Diseases; Assistant Radiologist, National Hospital. 2d edition. 406 pages; 287 illustrations. Oxford Medical Publications. Oxford University Press, New York, N. Y., publisher, 1951. Price $5.50.

The second edition of this text, revised after 16 years, reflects many years of experience. The subject matter covers the entire field of roentgenographic interpretation. Special emphasis is placed on the fine points of the diagnosis of the more common and frequently encountered pathologic conditions. Frequent mention is made of special and additional film views for demonstrating differential diagnostic points. The use of stereoscopic views is frequently stressed in bone and joint studies while in roentgenograms of the skull this technic is scarcely mentioned. The numerous and skillful pen-and-ink drawings by the author illustrate the subject matter well. It is regretted that no reproductions of x-ray films were used. The index is large and appears to be complete. The skeletal system has received the most comprehensive treatment. A chapter is devoted to each area with full discussion of the injuries, diseases, and tumors in order of relative frequency. Although this leads to a moderate amount of repetition, it gives a more complete differential picture. The alimentary tract is given almost as thorough consideration as in many special texts on the subject. The sections on head and brain, chest, genitourinary, and obstetrical measurements round it out into a good general reference. This book gives a nice blending of the British and American viewpoint. It is recommended for radiologists and clinicians alike.

—*Commander W. C. Mulry, MC, U. S. N.*

Varicella Complicated by Gangrene of the Lower Extremities[1]

Joseph W. Batch, *Colonel, MC, U. S. A.*

Samuel Sepkowitz, *Captain, MC, U. S. A.*

VARICELLA, a common contagious disease due to a filterable virus, is usually considered to be a mild disease without serious complications or sequelas. The case reported here indicates that serious complications may occur. Wishik and Bullowa (2) reported complications in 133 (5.2 percent) of 2,534 patients with varicella, 76 of whom suffered from infection of the surface of the body. In order of frequency the surface complications were: cutaneous abscess, lymphadenitis, cellulitis of the skin and subcutaneous tissue, erysipelas, gangrene of the skin and subcutaneous tissue, impetigo, paronychia, conjunctivitis, balanitis, hordeolum, blepharitis, stomatitis, and parotitis. Pemphigus gangraenosus was first referred to as a complication of varicella in 1807 by Whitney Stokes. Since that time several types of serious complications associated with varicella have been reported. Some of these include: malignant chickenpox (reported by Humphrey Storer in 1827), gangrenous varicella (3), bullous and gangrenous varicella (4), acute thrombocytopenic purpura following varicella (5), thrombocytopenic purpura with bleeding (6), thrombocytopenic

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(1) Army and Navy Hospital, Hot Springs, Ark.

(2) Wishik, S. M., and Bullowa, J. G. M.: Complications of varicella; surface complications. Am. J. Dis. Child. 49: 927-932, Apr. 1935.

(3) Barenberg, L. H., and Lewis, J. M.: Gangrenous varicella; case report. Arch. Pediat. 44: 653-656, Oct. 1927.

(4) Gordon, E.: Fatal case of bullous and gangrenous varicella. Arch. Pediat. 44: 337-341, June 1927; also M. Press 124: 176-178, Aug. 31, 1927.

(5) Cohen, H. J.: Acute thrombocytopenic purpura following varicella; report of case. Arch. Pediat. 53: 773-776, Dec. 1936.

(6) Coehn, J. J., and Bansmer, C.: Chicken pox with simultaneous idiopathic thrombocytopenic purpura; report of case. New England J. Med. 237: 222-224, Aug. 14, 1947.

purpura with gangrene (7), varicella and pemphigus acutus (8), and hemorrhagic chickenpox (9).

CASE REPORT

A 5-year-old girl was admitted to this hospital on 15 March 1951 because of gangrene of both lower extremities necessitating a bilateral amputation of the lower extremities. About 1 month prior to admission she developed a vesicular rash which was diagnosed chickenpox by the family physician. An older sibling had chickenpox about 2 weeks before. At the onset the patient's temperature was normal. The lesions were confined to the face and trunk and healed in a few days. One week after the onset large red blisters developed on both lower extremities. These new lesions began near the ankles as blisters about the size of an egg. They increased in size, progressing up the legs and covering both legs and buttocks in the course of a few hours. Beginning at the initial site, the lesions turned black as they progressed.

The patient became toxic, running a septic temperature with peaks up to 102° F., and refused to eat. Her family physician made a diagnosis of hemorrhage beneath the skin. Hot packs were applied, sedatives were given, and she was referred to a hospital in a nearby city. There, despite repeated blood transfusions and antibiotic therapy, she became progressively worse. On 28 February a bilateral fasciotomy of the legs was performed because it was believed that the progressive gangrene developing in the extremities was due to ischemia secondary to subcutaneous bleeding which caused pressure blockage of the deep vessels. No improvement followed this operation. Paravertebral blocks were performed daily for 1 week for the same reason and were thought to give temporary relief until 8 March. From this time on the condition became worse. On 14 March her legs were redressed under anesthesia and because amputation was deemed necessary the patient was transferred to this hospital.

Physical examination here revealed a child who was acutely ill, very irritable, toxic, and irrational at times, but who was co-operative when aroused. Her temperature was 103.8° F. Her pulse was 120. Her respirations were 30. Her blood pressure was 110/70. Her skin was dark and there were several small hyperpigmented areas on the trunk, apparently the residuals of the old varicellar lesions. There were numerous petechias on the left arm just below where a tourniquet had been applied. The lower extremities presented large irregular black eschars, which encircled both ankles and feet and extended up the lateral aspect of the legs and thighs to the buttocks. Along the lateral border of

(7) Stoesser, A. V., and Lockwood, W. W.: Varicella complicated with acute thrombocytopenic purpura and gangrene. J. Pediat. 12: 641-647, May 1938.

(8) Schwartzman, J.: Varicella and pemphigus acutus; report of case. Arch. Pediat. 57: 595-598, Sept. 1940.

(9) Ford, R. A., and Marin, R. B.: Hemorrhagic chicken-pox; case report. J. M. Soc. New Jersey 41: 440-441, Dec. 1944.

the thighs, legs, and feet were deep incisions which extended through the deep fascia and, in the legs, extended down to bone (fig. 1). Sensation was completely lost in the left leg and foot and partially in the right. Femoral pulsations were present bilaterally.

On admission the erythrocyte count was 4,620,000; the hemoglobin was 12.5 grams per 100 cc.; the leukocyte count was 16,800 with 60 percent neutrophils (18 percent were staff cells), 38 percent lymphocytes, and 2 percent eosinophils. The platelet count was 54,000. The clotting time was 3 1/4 minutes. The bleeding time was 1 minute. The

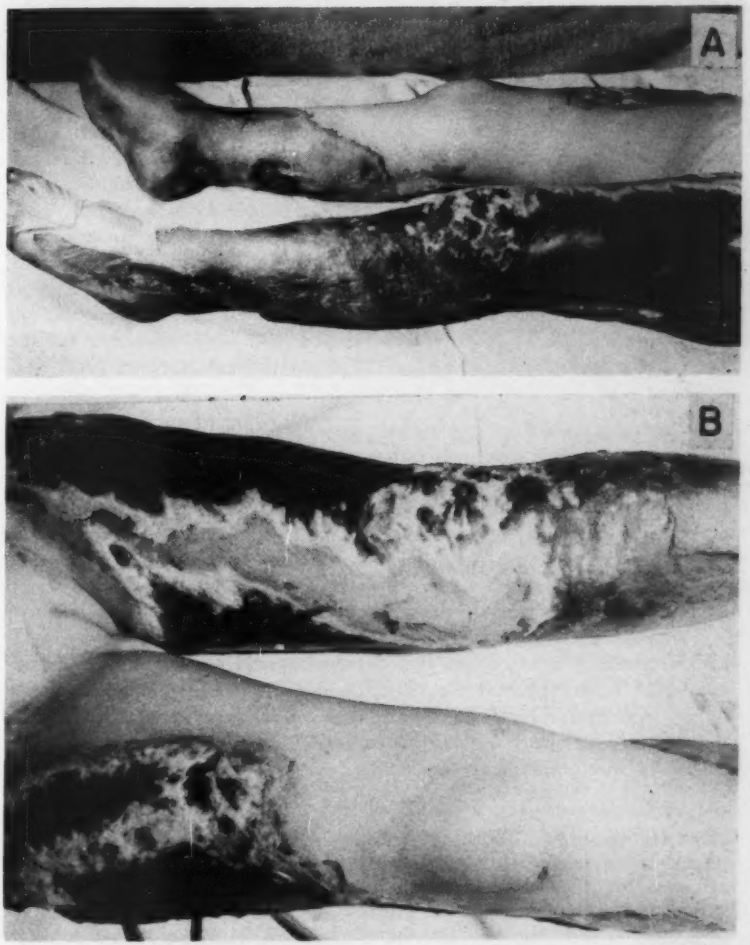

Figure 1. Photographs of lower extremities as they appeared on admission.

hematocrit was 47 percent. The sedimentation rate was 37. The prothrombin time was 23 seconds (control 15). The cephalin-cholesterol flocculation test was negative. Blood culture was negative. The tourniquet test was positive. *Proteus* and *Endamoeba coli* were cultured from the wounds on the leg (reported on 2 April).

On admission both of the patient's lower extremities were dressed under anesthesia and it was decided to treat her conservatively with débridement of the eschar, dressings, antibiotics *(10)*, and blood transfusions. She was given a high protein diet with vitamin supplements and 15 mg. of ACTH every 6 hours. On 17 March her lower extremities were dressed using crude penicillin. This was continued until 26 March when the crude penicillin dressings were supplanted by 10 percent aqueous mercurochrome and pressure dressings. Frequent dressings with moderate débridement were performed under anesthesia. On 31 March the wounds on her left leg had become sufficiently clean and the devitalized area of gangrene had become sufficiently demarcated so that an amputation through the middle third of the left leg could be performed. At operation it was noted that the anterior tibial artery was thrombosed. On 2 April it was apparent that the right leg could not be saved and it was amputated through the middle third. On 5 April, following amputation of both legs, split-thickness skin grafts were taken from available parts of the body and used to cover in part the granulating wounds of the buttocks, thighs, and legs. As areas became sufficiently clean, split-thickness skin grafts were applied until final coverage was obtained on 26 May.

As the infection was controlled, and particularly after the denuded areas were covered with split-thickness skin grafts, the patient began to show marked improvement. At the time of débridement of the wounds, representative sections of the eschar as well as the adjacent skin margin of more viable tissue were sent to the laboratory for study. The sections of the eschar revealed tissue which had completely lost its structure and only the outline of fatty tissue remained. There was a tremendous infiltration by erythrocytes. The tissue diagnosis was gangrene of the skin and subcutaneous tissue. The sections of skin margin revealed a small strip of fibrocollagenic tissue on the surface of which loose alveolar tissue permeated by numerous small hyperplastic capillaries was seen. This was infiltrated with numerous cells characteristic of chronic and acute inflammation. The histopathologic diagnosis was granulation tissue.

The patient was given whirlpool baths and exercises to strengthen the muscles of her lower extremities. The scar and skin grafts were softened by massaging them with lanolin. On 3 July she was allowed to spend the night at home with her parents. She was discharged from

(10) From 15 March through 9 June she was given 50,000 units of penicillin, 125 mg. of chloramphenicol, 125 mg. of aureomycin, and 0.25 mg. of streptomycin, each every 4 hr.

the hospital 17 August with a normal blood count and healed amputation stumps. The lesions of both legs had completely healed and the skin had become pliable. It was realized, however, that additional plastic operations would be required before the stumps would satisfactorily tolerate prostheses (fig. 2).

Figure 2. Photograph of leg stumps at time of discharge from hospital.

DISCUSSION

Cutaneous gangrene complicating chickenpox has been reported to occur only 4 times in over 2,454 cases observed by Wishik and Bullowa *(2)*. Usually the gangrene is associated with a thrombocytopenic purpura or a hemorrhagic diathesis. There is hemorrhage into the vesicles and into the cutaneous tissue as well as into other parts of the body *(11)*. These complications may begin at the height of the disease *(2-4)* which may be mild or severe at the onset. Hemorrhage and gangrene may develop after the lesions have crusted and started to clear. In the patient reported the disease was mild initially and complications did not develop until the seventh day of illness. The complicating lesions were not adjacent to or associated with the vesicles of varicella but were in a distant location.

Gangrene in this patient is best explained by a hemorrhagic diathesis developing after the onset of chickenpox. The large bluish areas, appearing suddenly and diagnosed by the family physician as hemorrhage beneath the skin, suggest a blood dyscrasia. The laboratory tests on this patient did not reveal any significant alterations in the bleeding and clotting mechanism, but since these determinations were made a month after the onset of disease, sufficient time had elapsed for any abnormal blood findings to return to normal *(5)*. An attempt to obtain laboratory data during the early phases of her illness was unavailing.

(11) Hoyne, A. L.: In McQuarrie, I. S. (editor): Brennemann's Practice of Pediatrics, Volume 2. W. F. Prior Co., Hagerstown, Md., 1948. chap. 3.

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ACTH was used in an effort to combat the toxic state and to reduce her fever *(12,13)*. Although her temperature was unaffected by 60 mg. of ACTH per day her general condition was much improved. She developed an excellent appetite, consuming a high protein, high caloric diet and frequently demanding extra portions of food. Clinically, evidence of toxicity was reduced, even at the height of the fever and the tissue destruction. The stupor and irrationality present on admission quickly disappeared.

From a therapeutic standpoint, the problem initially seemed to be not much different from that presented by gangrene from any cause or by a severe burn because the hemorrhagic tendency had abated when the patient was seen by us. The program was one of supportive therapy, control of infection and pain, and skin coverage. About 2 weeks might have been saved by earlier amputation of the lower extremities, but at that time it was deemed wiser to try to control infection by antibiotics and dressings and to determine the lowest level at which the remaining tissues would be viable. On this program initially some hope remained of saving at least one foot (and for a time it seemed as if such might be the case), but with the infection present, bone exposed without tissue to cover it, and the resultant gangrene and tissue necrosis it soon became obvious that we were hoping for more than could be obtained.

SUMMARY

In treating a patient with varicella complicated by a hemorrhagic condition resulting in gangrene of both lower extremities, the first objectives were control of infection and general supportive measures. Both legs had to be amputated and split-thickness skin grafts were used to cover the granulating wounds. General physical therapeutic measures were used during convalescence.

Complications of varicella are not common but may be severe even following a mild attack of the disease.

(12) Kass, E. H., and Finland, M. F.: Effect of ACTH on induced fever. New England J. Med. 243: 693-695, Nov. 2, 1950.

(13) Sprague, R. G.: Cortisone and ACTH; review of certain physiologic effects and their clinical implications. Am. J. Med. 10: 567-594, May 1951.

Blood Volume Estimation

Following the Infusion of Nonprotein Colloidal Solutions

Robert A. Phillips, *Commander, MC, U. S. N.* (1)

SHOCK due to a decrease in the circulating blood volume from hemorrhage, burns, diarrhea, et cetera can result in irreversible tissue damage and death. Treatment of this type of shock is facilitated by immediate restoration of the blood volume to the normal range. The large number of such casualties which would occur as a result of atomic warfare, points up the desirability of simple field methods for estimating blood volume. Previously (2) it has been shown that blood volume can be quickly estimated by determining whole blood specific gravities on 2 samples of blood, 1 drawn before and the other drawn 5 minutes after the rapid infusion of a known volume of plasma or albumin. The volume estimated is that which prevailed before the infusion. This report extends the usefulness of the procedure by pointing out that the technic, on the basis of other studies (3), is also applicable after dextran or other colloidal solutions are infused.

Technic. The specific gravity of whole blood is determined to ±0.0002 by the copper sulfate method (4) on samples of blood drawn immediately before and 5 minutes after the infusion of a known volume of colloidal solution of known specific gravity (5). It is not necessary to transfer the blood from the syringe to a tube containing an anticoagulant because drops of blood can be introduced into the copper sulfate solutions directly from the syringe's needle (4). The blood volume before infusion

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(1) With the U. S. Naval Medical Research Unit Number 3, Cario, Egypt, at time of writing this article; now with Research Division, Bureau of Medicine and Surgery, Department of the Navy, Washington, D. C.

(2) Phillips, R. A.; Yeomans, A.; Dole, V. P.; Farr, L. E.; and Van Slyke, D. D.: Estimation of blood volume from change in blood specific gravity following plasma infusion. J. Clin. Investigation 25: 261-269, Mar. 1946.

(3) Phillips, R. A.: Simple technic for following disappearance of infused colloidal solutions. To be published.

(4) Phillips, R. A.; Van Slyke, D. D.; Hamilton, P. B.; Dole, V. P.; Emerson, K., Jr.; and Archibald, R. M.: Measurement of specific gravities of whole blood and plasma by standard copper sulfate solutions. J. Biol. Chem. 183: 305-330, Mar. 1950.

(5) For use in the field it would be essential that the volume and specific gravity of the solution be stamped on the container.

can be quickly calculated from the nomogram (fig. 1). If the height of the patient is known, the nomogram also enables one to determine what percent of the expected normal volume this volume is.

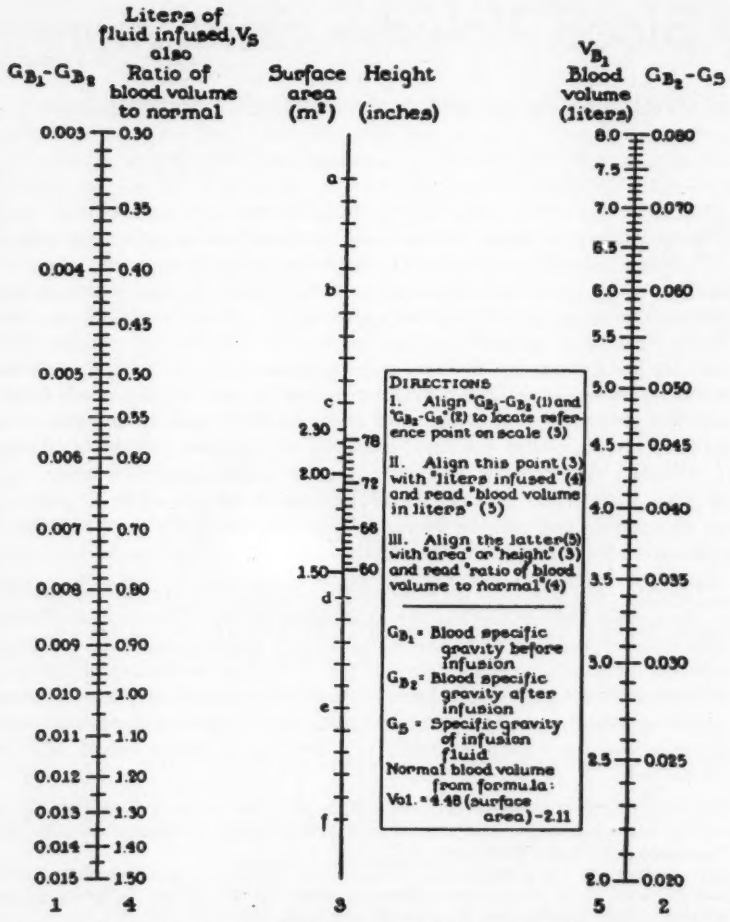

Figure 1. Calculation of blood volume from the change in blood gravity due to infusion of plasma or similar fluid. (From footnote reference (2).)

In vitro studies *(3)* have shown that whole blood specific gravity determinations by the copper sulfate method are accurate even when 1 volume of blood is mixed with 3 volumes of isotonic sodium chloride or 6 percent dextran. If an average man (surface area = 1.73 sq. m.), with a blood volume of 6,000 cc. *(2)*, loses 2/3 of his blood by hemorrhage, his blood volume would be 2,000 cc. The infusion of 6 liters

of dextran, 2 liters more than would be needed to restore his blood volume to normal, would result in a mixture of dextran and blood which has more than sufficient protein to insure an accurate reading of specific gravity by the copper sulfate method; hence it is evident that this method will determine whole blood specific gravities accurately under conditions of dilution which approach those at which life is impossible.

One lot of a solution of dextran gave blood volume values that averaged 13 percent less than the true blood volume *(3)*. It was shown that this was not an error in the technic of estimating the blood volume but was due to the rapid removal of 13 percent of the dissolved dextran. In consequence, for the technic to be useful under field conditions it would be necessary that the label of the solution's container state that the average loss of the colloidal substance 5 minutes after a rapid infusion into a normal subject will be x grams (and in consequence the blood volume estimations by the copper sulfate method will be y percent too low). This would not be necessary if the specifications for acceptance were sufficiently rigid so that the loss would not result in more than a 5 percent error.

Because the technic is applicable following plasma or albumin infusion *(2)* and following dextran infusion *(3)*, it will be equally useful following the infusion of known volumes of other plasma expanders, provided it is previously ascertained that a correction factor is required.

SUMMARY

A simple technic, applicable under field conditions, for estimating the blood volume following the infusion of a known volume of plasma has been found to be equally practicable when the infused material is dextran or other colloidal solutions.

BOOKS RECEIVED

Steps Toward Prevention of Chronic Disease, Summary of the National Conference on Chronic Disease (Preventive Aspects) Held March 12-14, 1951. Sponsor, *Commission on Chronic Illness.* Cosponsors, *U. S. Public Health Service, National Health Council.* 31 pages. Health Publications Institute, Inc., Raleigh, N. C., 1952. Price $.50.

Psychotherapy of Psychosis, by *Gustav Bychowski,* M. D., Assistant Clinical Professor of Psychiatry, New York University College of Medicine. 328 pages. Grune & Stratton, Inc., New York, N. Y., publisher, 1952. Price $5.75.

Transactions of the New York Academy of Sciences, January 1952. *Roy Waldo Miner,* Editor, and *James A. Goode,* Associate Editor. 144 pages. The New York Academy of Sciences, New York, N. Y., publisher, 1952. Price $.50.

Diagnostic and Experimental Methods in Tuberculosis, by *Henry Stuart Willis,* M. A., M. D., F. A. C. P., Superintendent and Medical Director, North Carolina Sanatoria, McCain, North Carolina; Consultant, United States Public Health Service; Formerly, Associate in Medicine, The Johns Hopkins University, and Visiting Physician, The Johns Hopkins Hospital, Baltimore, Md.; Formerly, Superintendent and Medical Director, Maybury Sanatorium, Northville, Mich., and *Martin Marc Cummings,* M. D., F. C. C. P., Director, Tuberculosis Research Laboratory, Lawson Veterans Administration Hospital, Veterans Administration, Chamblee, Ga.; Instructor in Medicine, Emory University School of Medicine, Atlanta, Ga.; Consultant, United States Public Health Service; Formerly, Director, Tuberculosis Evaluation Laboratory, Communicable Disease Center, United States Public Health Service, Atlanta, Ga. 2d edition. 373 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $10.

Developmental Disorders of Mentation and Cerebral Palsies, by *Clemens E. Benda,* M. D., Director of Research and Clinical Psychiatry, Walter E. Fernald State School, Waverley, Mass.; Instructor in Neuropathology, Harvard Medical School; Assistant in Psychiatry, Massachusetts General Hospital; Lecturer, Postgraduate Seminar, Massachusetts Department of Mental Health. 565 pages; 102 illustrations. Grune & Stratton, New York, N. Y., publisher, 1952.

A Manual for Psychiatric Case Study, by *Karl A. Menninger,* M. D. The Menninger Clinic Monograph Series No. 8. 355 pages; illustrated. Grune & Stratton, New York, N. Y., publisher, 1952. Price $6.75.

Urological Pathology, by *Peter A. Herbut,* M. D., Professor of Pathology, Jefferson Medical College and Director of Clinical Laboratories, Jefferson Medical College Hospital, Philadelphia, Pa. Two volumes. 1,247 pages; 527 illustrations, 2 in color. Lea & Febiger, Philadelphia, Pa., publisher, 1952. Price $24.

Cardiac Emergencies and Heart Failure, Prevention and Treatment, by *Arthur M. Master,* M. D., Cardiologist, Mount Sinai Hospital, New York, N. Y.; *Marvin Moser,* M. D., 1st Lt., USAF (MC), Walter Reed Army Hospital, Washington, D. C.; Former Fellow, Cardiology, Mount Sinai Hospital, New York, N. Y.; and *Harry L. Jaffe,* M. D., Adjunct Physician, Cardiology, Mount Sinai Hospital, New York, N. Y. 159 pages; illustrated. Lea & Febiger, Philadelphia, Pa., publisher, 1952. Price $3.

Shock Treatments, Psychosurgery, and Other Somatic Treatments in Psychiatry, by *Lothar B. Kalinowsky,* M. D., Research Associate in Psychiatry, College of Physicians and Surgeons, Columbia University, and New York State Psychiatric Institute and Hospital; Associate Attending Neurologist, Neurological Institute of New York, and *Paul H. Hoch,* M. D., Principal Research Scientist (Psychiatry), New York State Psychiatric Institute and Hospital; Assistant Professor, College of Physicians and Surgeons, Columbia University. Foreword by *Nolan D. C. Lewis,* M. D., Professor of Psychiatry, College of Physicians and Surgeons, Columbia University; Director of the New York State Psychiatric Institute and Hospital. 2d revised and enlarged edition. 396 pages. Grune & Stratton, New York, N. Y., publisher, 1952. Price $8.75.

Doctors in Blue, The Medical History of the Union Army in the Civil War, by *George Worthington Adams.* 253 pages; 16 illustrations. Henry Schuman, Inc., Publishers, New York, N. Y., 1952. Price $4.

Refraction and Motility, by *Walter B. Lancaster,* A. B., M. D., D. Sc. Hon. 310 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $7.75.

Fulminating Purpuric Meningococcemia[1]

Bruno Jastremski, *Colonel, MC, U. S. A.*

Jay C. Hoyt, *Captain, MC, U. S. A.*

BY 1947, 177 patients with the Waterhouse-Friderichsen syndrome had been reported *(2)*. Carey *(3)* reported the first case of recovery from adrenal hemorrhage with purpura and septicemia in 1940. Since then 19 cases have been added to the literature *(4-7)*. Most authors believe that it is the acute adrenal insufficiency which causes death in these patients, but some still maintain that death is due to toxemia. It is believed that the increased nonprotein nitrogen and serum potassium, with the drop in carbon dioxide combining power and sodium, as well as extreme shock are signs of adrenocortical insufficiency. Recent literature has demonstrated the value of cortisone in shock. Since 1940, many authors *(5-10)* have used or suggested the use of adrenocortical extracts in therapy, but as far as is known, no cases have been

(1) U. S. Army Hospital, Fort Leonard Wood, Mo.

(2) Wasserman, C. F.: Waterhouse-Friderichsen syndrome, with review of literature and addition of 3 new cases from Charity Hospital of Louisiana. New Orleans M. & S. J. 99: 286-293, Dec. 1946.

(3) Carey, T. N.: Adrenal hemorrhage with purpura and septicemia (Waterhouse Friderichsen syndrome) with recovery; case report. Ann. Int. Med. 13: 1740-1744, Mar. 1940.

(4) Christian, H. A.: Oxford Looseleaf Medicine. Oxford University Press, New York, N. Y. Vol. V, Part I, Chap. IV-A.

(5) Grubschmidt, H. A.; Graham, G. C.; and Jessup, E. C.: Case of fulminating meningococcemia exhibiting Waterhouse-Friderichsen syndrome and demonstrating value of cortical extract administration. Ann. Int. Med. 26: 294-304, Feb. 1947.

(6) Jacobi, M., and Harris, L.: Fulminating purpuric meningococcemia (Waterhouse-Friderichsen syndrome) with recovery. Ann. Int. Med. 22: 876-886, June 1945.

(7) Smith, M. H. D.: Am. J. Dis. Child. 69: 330-331, 1945.

(8) Taylor, C. E. (Pittsburgh), and Landry, V. E.: Waterhouse-Friderichsen syndrome; recovery from shock in fatal case. Ann. Int. Med. 26: 599-603, Apr. 1947.

(9) Thomas, H. B., and Leiphart, C. D.: Septicemia and purpura with adrenal hemorrhage in adult (Waterhouse-Friderichsen syndrome); discussion of role played by adrenal gland in production of syndrome; report of 2 adult cases. J. A. M. A. 125: 884-890, July 29, 1944.

(10) Kaufman, J. G.: Waterhouse-Friderichsen syndrome; report of case with recovery. J. M. Soc. New Jersey 41: 400-401, Nov. 1944.

reported in which cortisone has been used. We wish to report a patient with fulminating purpuric meningococcemia who survived apparently as a result of cortisone therapy.

CASE REPORT

A 22-year-old Negro was admitted to this hospital at 2000 hours on 13 February 1951. He had had an upper respiratory infection with rhinorrhea, malaise, and cough for 3 days prior to the onset of the present illness. Eight hours before admission he had a sudden chill and severe malaise. He went to bed and awakened at 1600 hours, feeling extremely weak. The last thing he could remember was going to the latrine. Between then and the time of admission, he vomited persistently, became delirious, and was unable to recognize his friends. There was no known exposure to meningitis.

On admission he was disoriented, hallucinated, restless, and uncooperative. His rectal temperature was 107° F.; his pulse was 140; his respirations were 30; and his blood pressure was 84/70. There was only slight nuchal rigidity. His pharynx was injected. Over his arms and the anterior surface of his chest there were several purple petechia which measured up to 4 mm. in diameter. The heart and lungs were within normal limits. The pupils were small, equal, and reacted to light. The reflexes were equal but hyperactive throughout.

The leukocyte count was 38,600 with 46 percent neutrophils, 20 percent stab forms, 24 percent lymphocytes, and 10 percent monocytes. The urine contained 2 plus albumin, 12 leukocytes per high-power field, and occasional hyaline casts. Blood culture revealed *Niesseria meningitidis.* The spinal fluid was clear, under normal tension, contained no cells, contained 87 mg. of protein per 100 cc., and was sterile on culture.

Immediately on admission the patient was given sodium amytal to quiet him so that intravenous medication could be given and was given 5 grams of sodium sulfadiazine in 2 doses 2 hours apart, followed by 1.5 grams every 4 hours, and 200,000 units of crystalline penicillin every 2 hours. A continuous intravenous infusion of 5 percent dextrose was maintained. That night the patient lapsed into coma with complete absence of all reflexes.

At 0700 on 14 February his rectal temperature had fallen to 101.2° F.; his pulse was barely perceptible with a rate of 120; his respirations were 30; and his blood pressure was 80/70. His bladder was distended and catheterization produced 1,000 cc. of clear urine. At 1000 on that day he appeared cyanotic. Fine rales were heard over all lung fields. Oxygen was administered by nasal catheter and 2 hours later his respirations were less labored, but the pulmonary edema persisted. His pulse was 140 and his blood pressure was 80/74. At 1400 hours his pulse and blood pressure were unobtainable. His circulating eosinophil count was 99. One hundred milligrams of cortisone were given and 4 hours later his circulating eosinophil count was zero. At 2200 his color was im-

proved; the pulmonary edema was still present; his pulse was feeble at 120 per minute; and his blood pressure was 72/56. A transfusion of 500 cc. of whole blood was given slowly, and his blood pressure held at 70/60 the rest of the night. His pharynx was aspirated repeatedly because of collection of mucopurulent exudate that interfered with the airway.

On the morning of 15 February, 32 hours after admission, his temperature reached normal. He coughed frequently, and required repeated aspiration. His pulse was stronger at 120 per minute, and his blood pressure ranged from 65 to 84 (systolic) and 50 to 70 (diastolic). His temperature rose to 100.4° F. that afternoon. Spinal puncture produced a thick purulent fluid under low pressure. Inadvertently this was not examined. He received another transfusion of 500 cc. whole blood. His color was better and his blood pressure was 84/65. The dose of cortisone was decreased to 50 mg. every 12 hours for the next 2 days.

At 0400 on 16 February, 54 hours after admission, he spoke for the first time. At 0700 his blood pressure was 84/68; his chest was clear to auscultation, the petechiae were less pronounced, and the patient was conscious, answering questions very sluggishly. Later that day he began to take fluids by mouth and by 2300 hours would respond to simple requests in appropriate fashion. On 17 February his blood pressure was 98/64. He expectorated blood-tinged sputum all that day, but on examination the heart and lungs were normal. He talked coherently and was able to take medication by mouth for the first time. Cortisone was discontinued and treatment with 2 grams of potassium acetate daily by mouth was begun. On 18 February his blood pressure was 90/67. He had begun to vomit when oral feedings and fluid were given, and supplements were given intravenously for the next 4 days. On 19 February his blood pressure reached 100/78. A roentgenogram of the chest was normal.

On 20 February he complained of slight pain in the calf of his left leg. Examination revealed good color, normal pulsations, reflexes, and temperature. On 21 February he again complained of a dull ache in the calf muscles. Again the physical findings were normal. At 1900 hours that day he complained of excruciating pain in the calf and inability to move the toes and left foot associated with numbness of the lower one-third of the left leg. Examination showed an extremity which was cold and blanched in its lower third. Dorsalis pedis and posterior tibial pulsations were absent on the left, but good pulsation was obtained in his left popliteal space. His right dorsalis pedis and posterior tibial pulsations were present. He could not move the toes of his left foot. Therapy consisted of immediate paravertebral block at the level of the first and second lumbar vertebras. This was followed by increased warmth of the affected area and ability to move the foot and toes. There was no decrease in pain. The patient was given 50 mg. of demerol for pain when needed, and was given heparin (by the intermittent intravenous injection technic) and dicumarol after the prothrombin and clotting times were found to be normal. No murmurs were ever heard. Clotting and prothrom-

bin times were held within a safe range. The extremity slowly improved until 23 February at which time only slight tenderness remained in the calf. At that time the patient developed some bleeding from his gums and petechiae on the skin. Anticoagulants were discontinued. His blood pressure was 100/80.

Cortisone therapy was resumed on 24 February and 10 mg. was given daily until 5 March when it was stopped. The patient showed steady

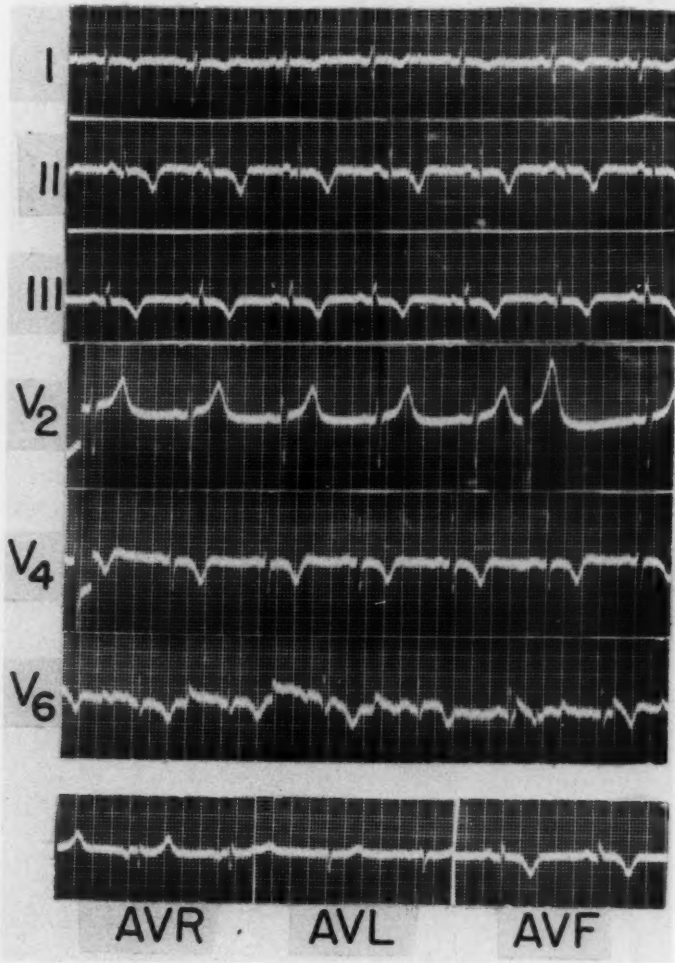

Figure 1. ECG taken on 5 March 1951.

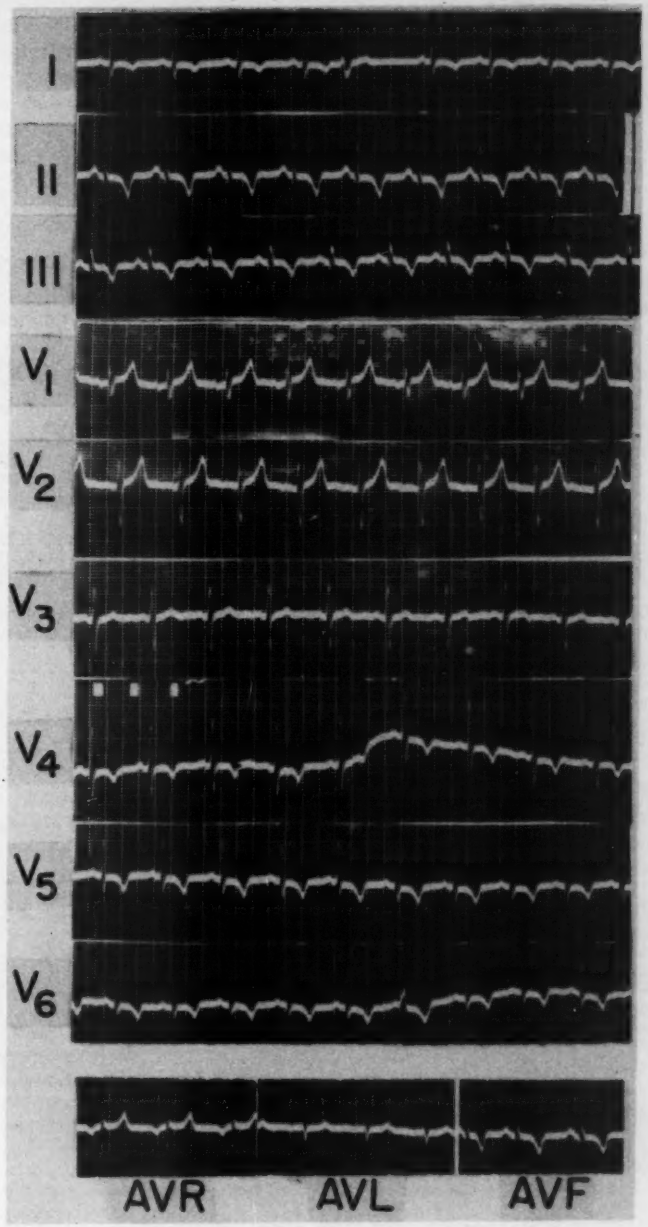

Figure 2. ECG taken on 11 April 1951.

clinical improvement. His highest diastolic pressure during this period was 106. Throughout the course the average daily intake of fluids was 4,000 cc. and the average output was 3,000 cc. On 28 February tender discolored areas of skin were observed under his left heel, and at the base of his left second toe. These were protected and did not slough nor become infected. On 3 March an ECG was taken, which showed deep inversion and coving of the T-wave in leads, I, II, III, AVF, V_3, V_4, V_5, and V_6 with an upright T-wave in lead AVR. It was believed that this was compatible with pericarditis. No friction rub had ever been heard in frequent auscultations by many observers. Roentgenograms of the chest on 19 February and 19 March showed a normal cardiac contour and size. Weekly ECG's have shown no change (figs. 1 and 2).

When the patient was last seen no pulsations could be felt in the left foot, but the temperature of the foot was normal. He still complained of numbness and coldness in the left foot on exposure to cold, but was otherwise asymptomatic. His blood pressure at that time was 104/80.

COMMENT

This case was diagnosed as fulminating purpuric meningococcemia on clinical grounds because of the combination of symptoms and signs associated with cyanosis, extreme shock, and coma. Although this syndrome was originally described as a pathologic condition consisting of hemorrhagic destruction of the adrenals in patients with septicemia, an extensive literature since 1940 has accepted it as a clinical entity. Many of these patients who meet the clinical criteria as outlined have shown no evidence of adrenal destruction. At the nadir of this patient's course, on the day following admission, his pulse and blood pressure could not be obtained. The circulating eosinophil count was 99. Following the administration of 100 mg. of cortisone the circulating eosinophil count dropped to zero and remained at zero on the following day. This may be interpreted as demonstrating adrenocortical deficiency in this patient. There was no suppression of kidney function despite his shock.

Septic arterial thrombi complicating the disease have been reported by Grubschmidt, Graham, and Jessup *(5)*. In view of the toxemia and hemorrhagic phenomena associated with this syndrome, we believe that the occlusion of the left popliteal artery in this patient might have resulted from an arteritis. The pericarditis noted is often seen in this syndrome.

ADDENDUM: Since this case report was submitted, an 11-year-old boy with Waterhouse-Friderichsen syndrome, successfully treated with cortisone, has been reported (Nelson, J., and Goldstein, N.: Nature of Waterhouse-Friderichsen syndrome; report of case with successful treatment with cortisone. J. A. M. A. 146: 1193-1197, July 28, 1951).

Preductal Coarctation of the Aorta

Case Report

Stuart H. Walker, *Major, MC, U. S. A.* (1)

THIS report of a case of preductal coarctation of the aorta clinically apparent in infancy demonstrates the ease with which the diagnosis of this potentially remediable congenital anomaly may be made. Calodney and Carson (2), in an excellent review of coarctation of the aorta in early infancy, reported 21 patients with this condition, 17 of whom were diagnosed ante mortem. Ante-mortem diagnosis of coarctation of the aorta had not previously been reported in infancy, yet the blood pressure differential between the upper and lower extremities which corroborated the diagnosis was readily obtainable in most patients in the series reported. The case herein reported was diagnosed chiefly through the discovery of a deep cyanosis of the lower trunk and legs with minimal cyanosis of the head and upper extremities, indicating a right to left (pulmonic to systemic) shunt into the aorta below the origin of the great vessels to the head and upper extremities. This type of cyanosis is pathognomonic of a preductal coarctation.

Surgical correction of coarctation of the aorta has been accomplished by several technics since first introduced by Gross and Hufnagel (3) in 1945. Only rarely, however, has correction been attempted in early infancy, and to my knowledge, never successfully. This situation is chiefly due to the infrequency of the ante-mortem diagnosis of the lesion and to the presence of advanced cardiac failure by the time the diagnosis is made rather than to a lesser amenability to operation of those lesions becoming clinically apparent in infancy. Calodney and Carson suggested that in the correction of coarctation in infancy the use of arterial grafts will usually be feasible although direct anastomosis or the production of a bypass through the left subclavian artery may

(1) William Beaumont Army Hospital, Fort Bliss, Tex.

(2) Calodney, M. M., and Carson, M. J.: Coarctation of aorta in early infancy. J. Pediat. 37: 46-77, July 1950.

(3) Gross, R. E., and Hufnagel, C. A.: Coarctation of aorta; experimental studies regarding its surgical correction. New England J. Med. 233: 287-293, Sept. 6, 1945.

be difficult to achieve. They recommended that every symptomatic case detected in infancy be subjected to a remedial operation as soon as cardiac failure and/or associated illnesses have been corrected.

CASE REPORT

The patient, a white male infant, was considered normal at birth. He gained well and showed normal behavior patterns until 2 months of age when his mother noted a persistent and progressively more severe dry cough. Listlessness and progressive weakness became increasingly apparent until marked difficulty in sucking was noted. The infant appeared dyspneic, being unable to suck for more than a few seconds at a time. When he was about 11 weeks old his mother first noted cyanosis associated with crying and feeding. These symptoms increased in severity and he was admitted to the hospital at 3 months of age.

Physical examination on admission revealed a fairly well-developed and nourished infant in moderate respiratory distress with moderate generalized cyanosis apparent on crying. Examination of the chest wall revealed a bulge along the left sternal margin maximal in the third left intercostal space, diminished breath sounds over the lower lobe of the left lung posteriorly, and increased cardiac dullness extending 1 cm. to the right of the sternum and to the left midaxillary line. Auscultation of the heart revealed sounds of fair quality, regular rhythm, a pulse rate of 160, and no murmurs. The liver was palpable 5 cm. below the right costal margin in the nipple line and was moderately tender. A marked cyanosis of the lower extremities was apparent with minimal cyanosis of the head and upper extremities. This was particularly noticeable on comparing the toe and fingernail beds. Peripheral pulses, including the femoral, were normal. Because infant-sized blood pressure cuffs were not available, no accurate blood pressure readings could be obtained.

Radiologic examination of the chest revealed marked cardiac enlargement, apparently of all chambers, the left side of the heart entirely filling the left lower portion of the chest. Moderate increase in hilar markings, thought to indicate pulmonary congestion, was apparent, but the periphery of all lobes revealed decreased vascular markings, thought to indicate reduced pulmonary blood flow. Electrocardiographic examination revealed abnormal right axis deviation with right ventricular preponderance, indicated by tall R waves and small S waves in leads aVR, V_1, V_2, V_3, V_4, and deep S waves in leads V_5 and V_6. An auricular tachycardia was suspected because of the regular rhythm, a rate of 167, and the presence of abnormally high-peaked P waves in lead II. Elevation of the S-T segment across the left precordium and depression of this segment across the right indicated myocardial injury.

A diagnosis of preductal coarctation of the aorta, based on the differential cyanosis in feet and hands, the evidence of right ventricular

hypertrophy and dilatation, the apparent decrease in pulmonary blood flow, and the appearance of cardiac failure between the second and third month of life was made. Digitalization with 0.03 mg. of lanatoside C intravenously per kilogram of body weight to correct the cardiac failure and stop the auricular tachycardia was attempted. The tachycardia, unaltered by several attempts at vagal stimulation, continued.

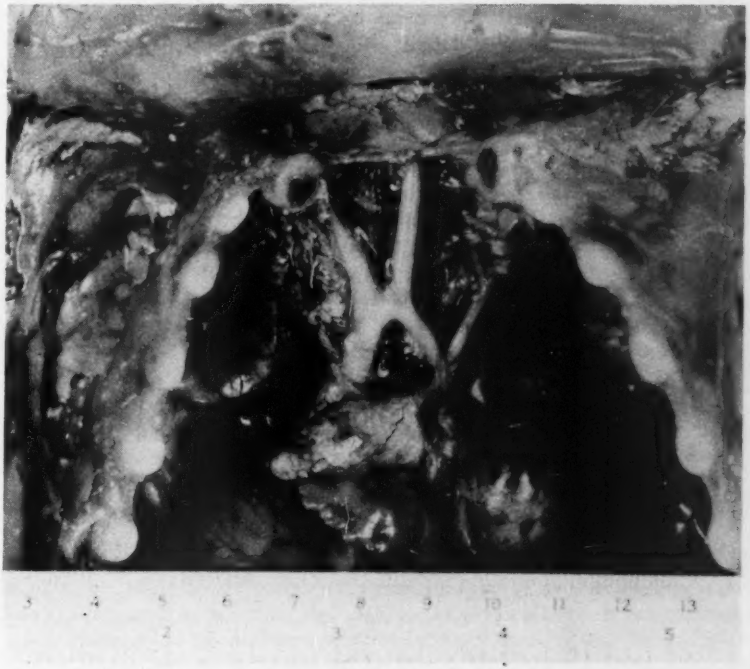

Figure 1. Photograph of necropsy specimen showing coarctation of the aorta immediately proximal to the entrance of the ductus arteriosus.

Though it was hoped that a return to normal rhythm would permit restoration of cardiac compensation and possibly eventual surgical correction of the lesion, this could not be achieved. A progressive rise in temperature with increased dyspnea and lethargy preceded the slowing and eventual cessation of cardiac contractions 20 hours after admission.

Necropsy revealed hepatomegaly due to congestion (the liver weighed 174 gm.); generalized pulmonary atelectasis, particularly in the lingula pulmonis; and displacement of the diaphragm and both lungs by massive

cardiac enlargement (the cardiothoracic ratio was 8.5/11). The heart weighed 103 gm. and was enlarged in all diameters; the thickness of the right ventricular wall was 0.9 cm. compared to 1.2 cm. for the left. No abnormalities of the heart or of the origin of the great vessels were detected. An abrupt, short constriction of the aorta was discovered distal to the origin of the left common carotid artery and just proximal to the ductus arteriosus and the left subclavian artery (fig. 1). The ductus arteriosus was widely patent. The constriction appeared to be a medial fibrosis without change in the intima. The circumference of the aorta was 0.7 cm. at the point of constriction and 1.2 cm. proximal and distal to this point.

COMMENT

In addition to the usual simple division into infantile and adult types *(4)*, a classification of coarctation of the aorta clinically apparent in infancy may be made for the purpose of consideration for surgical correction. A group with preductal (infantile) or possibly postductal (adult) coarctation so marked that death occurs in the immediate neonatal period may be distinguished as not amenable to operation. A second group of patients with preductal coarctation of a lesser degree which is compatible with life for from 1 to several months may be distinguished as distinctly amenable to and in dire need of operation. A third group which includes patients with severe postductal obstruction sufficient to produce symptoms in early infancy is also considered to be in need of early operation. Most clinically apparent cases in infancy fall into the second group and constitute a large portion of all cases of cardiac failure in this age group *(5)*. This group, into which the case reported falls, consists of patients apparently normal at birth, first demonstrating such signs of cardiac failure as weakness, dyspnea, cyanosis, cardiac and hepatic enlargement, and edema between 1 week and several months of age. They show evidence by physical, electrocardiographic, and roentgenographic examination of right ventricular hypertrophy and dilatation (as well as left ventricular hypertrophy) and characteristically have a lower blood pressure in the lower than in the upper extremities despite the usual presence of femoral artery pulsations. They occasionally demonstrate the characteristic differential extremity cyanosis, noted in the case reported herein, due to the shunt of venous blood through the patent ductus arteriosus into the area of decreased pressure in the aorta distal to the coarctation. This condition has apparently not been noted in previously reported cases diagnosed ante mortem.

The case reported showed a simple sharp coarctation immediately proximal to the entrance of the ductus arteriosus which would readily have lent itself to surgical correction by a variety of means. No other

(4) Taussig, H. B.: Congenital Malformations of Heart. Commonwealth Fund, New York, N. Y., 1947.

(5) Bahn, R. C.; Edwards, J. E.; and DuShane, J. W.: Coarctation of aorta as cause of death in early infancy. Pediatrics 8: 192-203, Aug. 1951.

major defects were discovered which would have prevented surgical cure. Calodney and Carson *(2)* stated that only 27 percent of their series of 21 patients, chiefly of this second group, had major associated anomalies which would have negated the value of surgical correction. If this patient had been seen earlier (at which time the diagnosis should have been equally apparent), it is quite possible that a successful operation could have been performed.

The occurrence of a blood pressure differential between the upper and lower extremities in every patient with coarctation of the aorta, regardless of type, and the occasional presence of the pathognomic differential cyanosis in the second group indicate the ease with which the surgically correctable lesions may be diagnosed if they are considered. There can be little doubt that rapid advances in cardiac surgery will soon permit the successful correction of these anomalies if the clinician will but diagnose them in time.

BOOKS RECEIVED

Lippincott's Handbook of Dental Practice, edited by *Louis I. Grossman,* D. D. S., D. M. D. Authors: *W. Harry Archer,* M. S., D. D. S.; *Carlisle C. Bastian,* D. D. S.; *Basil G. Bibby,* B. D. S., Ph. D., D. M. D.; *Allan G. Brodie,* D. D. S., Ph. D.; *Lester W. Burket,* D. D. S., M. D.; *James R. Cameron,* D. D. S.; *Edward C. Dobbs,* D. D. S.; *Edward M. Grevatt,* D. D. S.; *Louis I. Grossman,* D. D. S., D. M. D.; *A. O. Klaffenbach,* D. D. S.; *Benjamin Kornfeld,* D. D. S.; *Howard K. Mathews,* D. D. S.; *Balint Orban,* M. D., D. D. S.; *Edward C. Stafne,* D. D. S.; *J. J. Stetzer, Jr.,* D. D. S.; *Charles A. Sweet,* D. D. S.; *V. R. Trapozzano,* D. D. S.; *Joseph F. Volker,* D. D. S., Ph. D.; and *Gordon R. Winter,* D. D. S. 2d edition. 503 pages; 388 illustrations; 4 color plates. J. B. Lippincott Co., Philadelphia, Pa., publisher, 1952. Price $10.

Diagnostic Bacteriology, A Textbook for the Isolation and Identification of Pathogenic Bacteria, by *Isabelle Gilbert Schaub,* A. B., Technical Director, Clinical Bacteriology Laboratories, The Johns Hopkins Hospital; Instructor in Bacteriology, The Johns Hopkins University School of Medicine and the Nurses Training Schools, The Johns Hopkins Hospital and Sinai Hospital, and *M. Kathleen Foley,* M. A., Instructor in Bacteriology, Department of Biological Sciences, College of Notre Dame of Maryland; Formerly Bacteriologist in Charge of the Diagnostic Bacteriological Laboratory of the Medical Clinic, The Johns Hopkins Hospital. 4th edition. 356 pages. The C. V. Mosby Co., St. Louis, Mo., publisher, 1952. Price $4.50.

A Textbook of Clinical Neurology, With an Introduction to the History of Neurology, by *Israel S. Wechsler,* M. D., Clinical Professor of Neurology, Columbia University, New York; Consulting Neurologist, The Mount Sinai Hospital, Montefiore Hospital and Rockland State Hospital, New York. 7th edition. 801 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1952.

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Signs and Symptoms, Applied Pathologic Physiology and Clinical Interpretation, edited by *Cyril Mitchell MacBryde,* A. B., M. D., F. A. C. P., Associate Professor of Clinical Medicine, Washington University School of Medicine; Assistant Physician, the Barnes Hospital; Director, Metabolism and Endocrine Clinics, Washington University Clinics, St. Louis, Mo. 2d edition. 783 pages; 98 illustrations; 50 charts; 8 color plates. J. B. Lippincott Co., Philadelphia, Pa., publisher, 1952. Price $10.

The 1951 Year Book of Urology (November 1950-October 1951), edited by *William Wallace Scott,* M. D., Ph. D., Director, James Buchanan Brady Urological Institute, Johns Hopkins Hospital; Urologist-in-Charge, Johns Hopkins Hospital; Professor of Urology, Johns Hopkins University School of Medicine. 383 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1952. Price $5.50.

Textbook of Clinical Parasitology, by *David L. Belding,* M. D., Professor of Bacteriology and Experimental Pathology, Emeritus, Boston University School of Medicine. 2d edition. 1,139 pages; illustrated. Appelton-Century-Crofts, Inc., New York, N. Y., publisher, 1952. Price $12.

BOOK REVIEW

Post-Graduate Lectures on Orthopedic Diagnosis and Indications, by *Arthur Steindler,* M. D., F. A. C. S., Professor of Orthopedic Surgery, State University of Iowa, Iowa City, Iowa. Volume III, Section A: Tuberculosis of the Skeletal System; Section B: Osteomyelitis. 284 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $8.75.

The conception that tuberculosis is a general systemic disease and skeletal tuberculosis merely one of its local manifestations is in agreement with the internist's point of view as presented in this series of lectures. The subject of tuberculosis of the skeletal system is well presented by first describing the general problems and treatment and then, in detail, the treatment of the individual joints and bones that may be involved. In each lecture the pathologic changes, clinical signs, diagnosis, and treatment are discussed. The second section of this book deals with osteomyelitis. The author emphasizes the fact that although antibiotics are now used in the treatment of acute and chronic osteomyelitis, the old surgical principles still hold true, namely, the development of natural resistance and immunity. The treatment of the localized lesion must be dealt with on surgical principles of evacuation and drainage and the spreading of local lesion controlled by immobilization.—*Lt. Col. J. R. Vivas, MC, U. S. A.* and *Col. R. E. Reiner, MC, U. S. A.*

False Aneurysm of the Facial Artery[1]

Leonard Petitti, *Lieutenant Colonel, MC, U. S. A.*

Hal B. Jennings, Jr., *Lieutenant Colonel, MC, U. S. A.*

THE purpose of this article is to report an unusual case of a false aneurysm of the facial artery resulting from a small penetrating wound of the face. We believe this to be the first of its kind to be reported. In our review of the literature we found only 2 cases of aneurysms about the face. One involved the temporal artery and resulted from a blow to the temple (2); the other involved the facial artery and resulted from the application of a Roger Anderson pin (3). The latter was more of an arteriovenous fistula than a false aneurysm.

CASE REPORT

On 4 October 1951 in Korea the patient suffered a shell-fragment wound of the left eye, a grazing wound of the left shoulder, and a small penetrating wound just above the left corner of the mouth. On the following day he noticed a small pulsating lump at the point of the wound of the upper lip (fig. 1a). A dressing was placed over it and no treatment given. Except for the pulsating sensation in the lump, the patient had no other symptoms. He was evacuated by air to the United States and on 12 October, while in flight, a nurse, thinking that the mass was accumulated blood, attempted to squeeze it out. Following this, the blood squirted across the plane and the patient bled profusely. A pressure dressing was applied in order to check the bleeding. The patient was admitted to this hospital on 15 October with a pulsating mass about the size of a walnut just above the corner of the mouth at the left nasolabial fold. The skin at the apex of the mass was denuded in a small area and a blue sac from which fresh blood oozed could be seen beneath this. The pulsation could be palpated and seen both over

(1) From the Brooke Army Hospital, Fort Sam Houston, Tex.

(2) Tippett, G. O.: Two cases of traumatic aneurysm. Brit. M. J. 2: 711-712, Oct. 15, 1932.

(3) Greeley, P. W., and Throndson, A. H.: Arteriovenous aneurysm resulting from application of Roger Anderson splint. J. A. M. A. 124: 1128, Apr. 15, 1944.

the wound and in the oral cavity. Roentgenograms of the head revealed a small metallic foreign body in the soft tissue of the mass.

The patient was taken to the operating room for removal of the mass. Two curved Kelly clamps, with the ends protected by rubber tubing and with rubber bands on the shanks, were placed above and below the lesion to insure hemostasis during the operation (fig. 1b). An

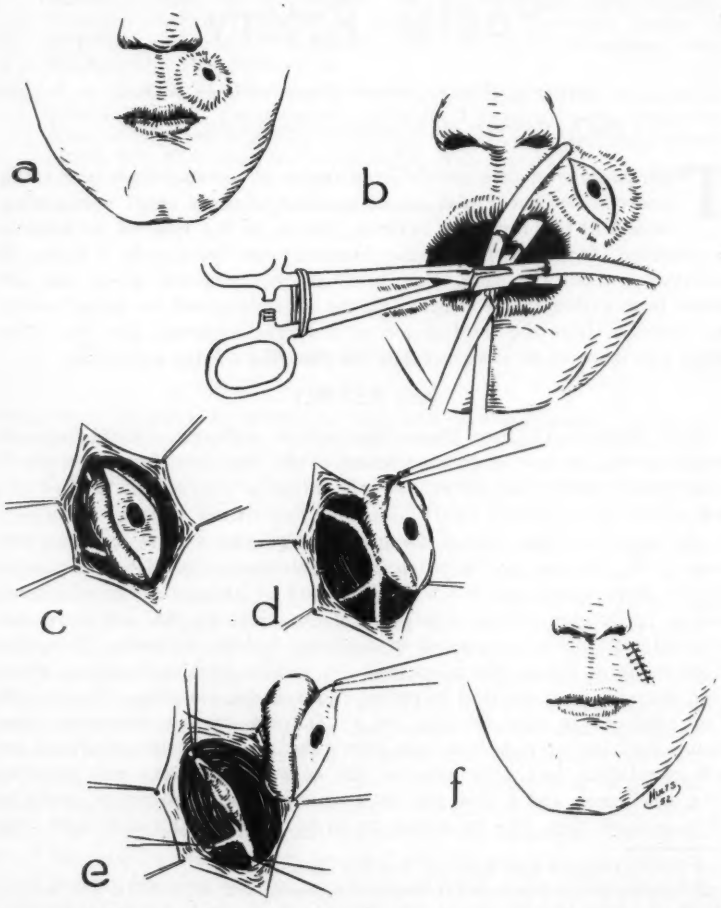

Figure 1. (a) *Sketch showing location of pulsating mass of upper lip and small perforated wound.* (b) *Hemostatic clamps in place.* (c) *Sac in place showing its bed and a definitely formed wall.* (d) *Sac removed from bed and showing attachment to the underlying tear in the facial artery.* (e) *Linear tear in the artery.* (f) *Postoperative appearance of wound.*

elliptical incision through the skin was made over the top of the mass and by means of sharp and blunt dissection, the sac was freed from the surrounding tissue (fig. 1 c, d, and e). The dissection was carried out until the base of the sac was reached. A small metallic foreign body was found lodged at the base and outside of the sac. After completely freeing the sac, it was lifted from its bed. The base was attached to the facial artery, extending from a point where it gives off the superior labial artery to 1 cm. along its course as it becomes the angular artery. Its wall was continuous with the arterial wall. After removing the sac, a tear in the artery 1 cm. long was seen. The artery was freed and ligated above and below the tear. The postoperative course was uneventful.

DISCUSSION

The terms false aneurysms and pulsating hematoma are used interchangeably in the literature *(4)* and designate any arterial tear which results in a blood- or clot-filled sac into which an artery continually pours blood. The enclosure may consist of not more than a partially organized clot bound by muscle, fascia, or skin surfaces or it may be a firm, fibrous, well-organized wall, depending on the site and duration of the lesion. False aneurysms in the region of the face are uncommon probably because the arteries about the face are of small caliber. There are numerous reports of false aneurysms occurring in penetrating wounds of the extremities and about the neck, where the arteries are larger and the fascial planes are more definite thus facilitating hematoma formation. They may be caused by a contusion, a knife or bullet wound, small pieces of shell fragments, small pieces of broken bone, or even venipuncture.

Following a tear in an artery, there is extravasation of blood into the surrounding area. If the conditions are right, the blood accumulates locally and does not extravasate to distant areas through fascial planes. A clot is formed around the arterial wound and as time goes on, the central portion of the clot is hollowed out by the pressure transmitted through the arterial pulsations. Fibroblastic proliferation occurs at the periphery. Organization continues until a fibrous wall is formed. The central space continues to enlarge and its inner lining becomes continuous with the endothelium of the artery.

False aneurysms are rare because:

1. As a rule, when a large artery is pierced, its companion vein is also injured, resulting in the formation of an arteriovenous fistula rather than a false aneurysm.

2. If there is complete severance of an artery, the ends of the severed artery retract and although a clot forms locally, the contiguity of blood

(4) Beck, W. C.: "Pulsating" hematoma (false aneurysm). War Med. 4: 502-507, Nov. 1943.

flow is broken and conditions are not favorable for the development of a false sac.

3. If a large artery is involved and bleeding persists, a surgeon will investigate before enough time for the formation of a false sac elapses.

4. The conditions necessary for the development of a false aneurysm are themselves rare.

These conditions are (1) the presence of a small penetrating wound of the arterial wall; (2) local extravasation of blood; (3) enough local pressure from surrounding fascial planes, muscles, or skin to limit clot formation around the arterial defect; (4) enough time to permit liquefaction and organization of the clot; and (5) continued patency of the arterial defect.

BOOK REVIEW

Essentials of Histology, by *Margaret M. Hoskins*, Ph. D., and *Gerrit Bevelander*, Ph. D., New York University. 2d edition. 240 pages; 135 illustrations; 2 color plates. The C. V. Mosby Co., St. Louis, Mo., publisher, 1952. Price $4.

In this new edition, the author has presented the morphologic characteristics of tissues and organs in concise language aided by drawings and diagrams. The book is divided into 17 chapters, each of which deals with a single anatomic system or set of organs. The only adverse statement that can be made is that the drawings are chiefly of the stippled type and hence not adequately detailed, as compared with those appearing in larger texts on this subject. This book is, however, well suited to students of elementary histology.

—*Commander J. S. Shaver, MC, U. S. N.*

P-Wave Inversion

Kenneth P. Bachman, *Lieutenant Commander, MC, U. S. N.*

ELECTROCARDIOGRAPHIC recording of the electrical impulses resulting from activity of the auricular musculature results in the visualization of a wave commonly termed the P wave. Rarely an auricular T wave may be seen. Although the P wave may normally be inverted in lead 3 of the routine ECG and frequently is inverted in lead 2 as a result of A. V. nodal rhythm, P-wave inversion is rarely seen in lead 1. Furthermore, some basic textbooks on electrocardiography fail to make note of such P-wave inversion in lead 1. Vakil and Bharucha *(1)* in what was at the time considered to be a comprehensive review of P-wave abnormalities also failed to note this rarely seen abnormality, although more recently P_1 inversion has been observed in patients with acute rheumatic fever *(2, 3)*.

The following case is unusual in that the patient was a 37-year-old healthy man who was thoroughly examined, including x-ray and laboratory studies, and who was found qualified for arduous physical duties. His initial tracing (fig. 1) revealed P-wave inversion in leads 1, 2, 3, and aV_F, an upright P wave in lead aV_R, and a variable essentially isoelectric P wave in lead aV_L. In the precordial leads the P waves may be noted to be upright in leads V_1, V_2, and V_3. The P-R interval was 0.16 seconds. At the time this tracing was taken the patient was moderately apprehensive.

One week later, with the patient fully relaxed, tracings taken in the supine, the sitting, and the left and right lateral positions revealed normal upright P waves in leads 1, 2, 3, aV_L, and aV_F (fig. 2). The P-R interval had lengthened to 0.2 seconds. Carotid sinus pressure, attempted first on the right and then on the left side, initially produced no result. The patient was purposely made apprehensive. During this procedure a direct writer was continuously recording lead 1. P_1 remained upright, with the patient in an apprehensive state, until right

(1) Vakil, R. J., and Bharucha, E. P.: Auricular wave; exhaustive inquiry into physiological and pathological variations of the P wave (or auricular wave) of electrocardiogram. Indian Physician. 5: 1, Jan. 1946; 32, Feb. 1946; 82, Apr. 1946.

(2) Filberbaum, M. B.; Griffith, G. C.; Solley, R. F.; and Leake, W. H.: Electrocardiographic abnormalities in 6,000 cases of rheumatic fever. California & West. Med. 64: 340-346, June 1946.

(3) Sampson, J. J., and Caplan, P. S.: Alteration of electrocardiographic P waves in acute rheumatic fever. Am. J. M. Sc. 212: 321-326, Sept. 1946.

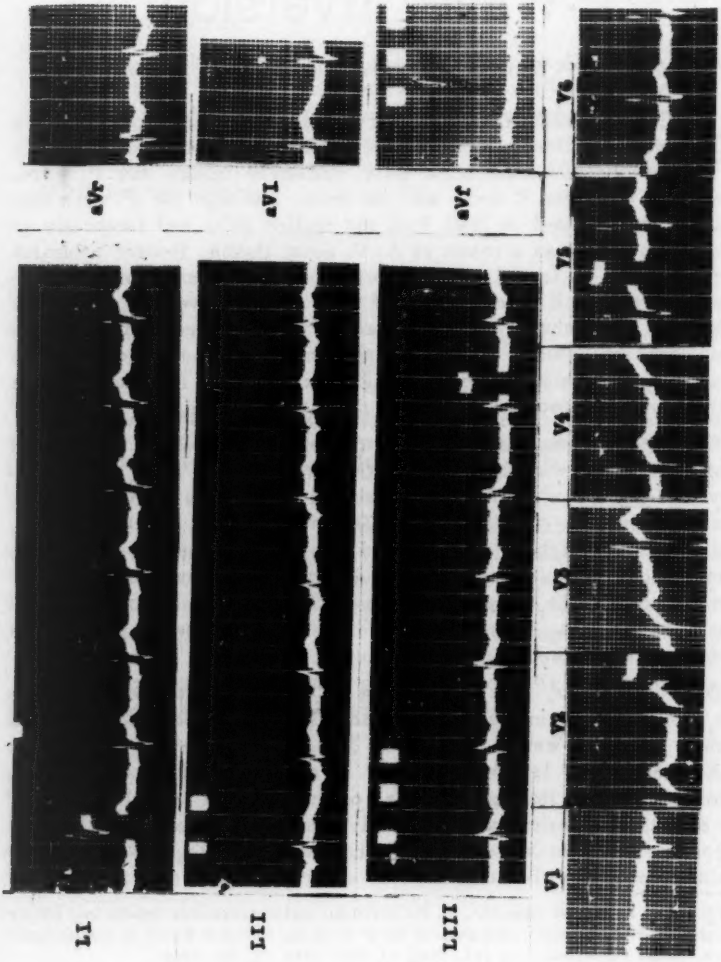

Figure 1.

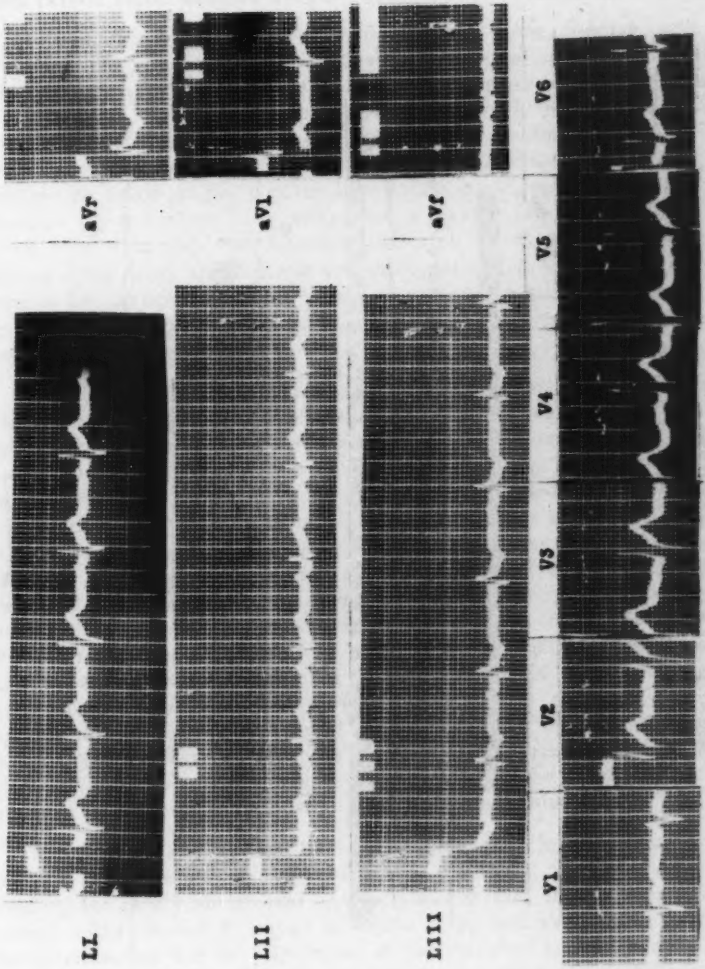

Figure 2.

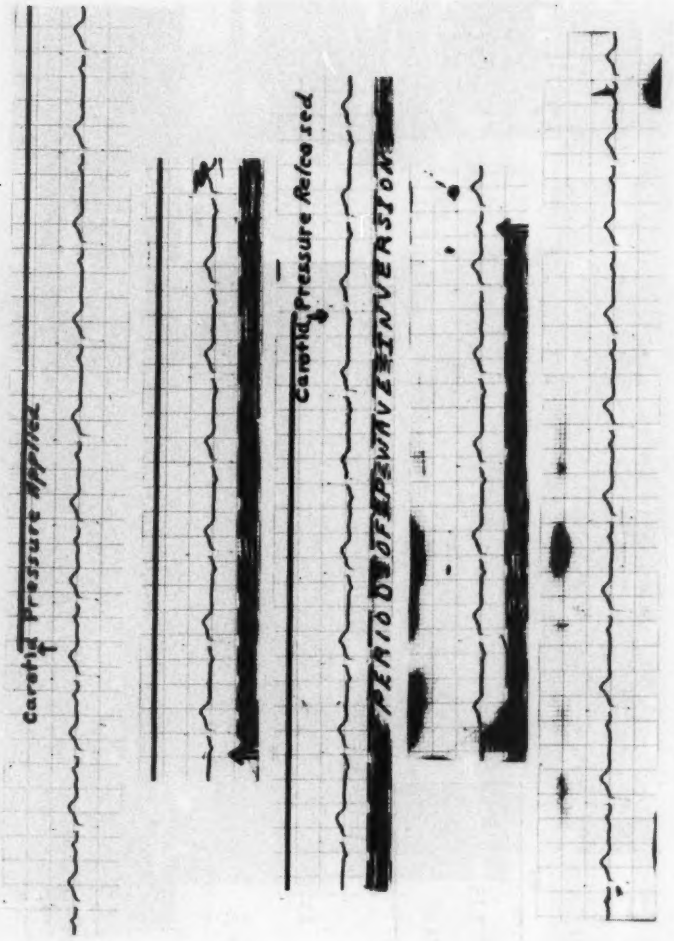
Carotid Pressure Applied
Carotid Pressure Released
PERIOD OF EXPERIMENTATION

Figure 3.

carotid sinus pressure was applied. As may be seen in figure 3 such pressure shortly resulted in the inversion of the P waves as previously noted in figure 1. With release of the right carotid sinus pressure the P waves, within 9 complexes, returned to the normal upright position. Left carotid sinus pressure did not cause any alteration of the P waves.

Whereas, the normal P-wave impulse passes down, forward, and to the left, the aforenoted type of P-wave inversion indicates that the electrical impulses as represented by the P waves were passing upward, forward, and to the right. Admittedly the P_2 and P_3 inversion, even with a P-R interval of 0.16 seconds, could be due to a high A. V. nodal rhythm, particularly if A. V. nodal conduction was prolonged (below the origin of the impulse high in the A. V. node). Furthermore, Sokolow *(4)* and others have stated that P-wave inversion in lead aV_F, even with a normal P-R interval, indicates an A. V. nodal rhythm. On the other hand, although the possibility of a high A. V. nodal rhythm cannot be excluded, several alternative explanations are believed to be more acceptable, particularly in this case with P_1 inversion in a normal healthy person.

The most acceptable explanation, I believe, is as follows: The patient has within his sinus node, which may be unusually long (the normal length is from 25 to 50 mm.), what may be considered to be a double pacemaker, or he has islands of sinoauricular (S. A.) nodal tissue or ramifications of the S. A. node in the neighborhood of the coronary sinus. In either event his usual pacemaker lies high in the S. A. node. As a result of vagal stimulation the pacemaker shifts to the lower part of the S. A. node, possibly some part lying fairly close to the coronary sinus. The location of the latter pacemaker in conjunction with the position of the heart (figs. 4-6) could fully account for all the unusual P-wave inversion. This patient's heart was electrically semihorizontal and in roentgenograms appeared anatomically even more horizontal. More important, it was rotated clockwise about its own long axis so as to make the pulmonary arterial segment (as seen in figure 1) appear unusually prominent for this type of heart. This unusual cardiac position is believed to be primarily responsible for the inversion of P_1 because the pacemaker is located near the coronary sinus.

The work of Ruskin and Decherd *(5)* tends to indicate that even the so-called coronary-sinus rhythm may not be a true A. V. nodal rhythm, but merely an unusual type of sinus rhythm, in that it is associated with inverted P waves in leads 2, 3, and aV_F, such P-wave inversion being due to origination of the impulse in "(1) islands of S. A. nodal tissue or ramifications of the S. A. node in the neighborhood of the coronary sinus; (2) in the tail of the S. A. node, nearer the A. V. node than normal; or (3) in the usual position in the S. A. node,

(4) Sokolow, M.: Clinical value of unipolar extremity (aV) leads. Ann. Int. Med. 34: 921-947, Apr. 1951.

(5) Ruskin, A., and Decherd, G. M.: Momentary atrial electrical axes; A-V nodal rhythm. Am. Heart J. 29: 633-641, May 1945.

Figure 4. Posteroanterior roentgenogram of chest. Figure 5. Right anterior oblique roentgenogram of chest. Figure 6. Left anterior oblique roentgenogram of chest.

but with unusually rapid conduction (a) from the S. A. node to the A. V. node, possibly through special functional pathways, or (b) through the A. V. node."

Filberbaum et al. *(2)*, in 6,000 patients with rheumatic fever with electrocardiographic changes, found 98 whose tracings revealed P-wave abnormalities. Of these, 9 were said to reveal inverted P waves in lead 1. The single picture of inverted P waves as printed in their article, however, revealed retrograde P waves appearing after the QRS complexes followed by dropped beats. They stated that "inversion of the P waves in leads 1 and 2 is probably a result of a shift of the site of the pacemaker in and away from the sinoauricular node." The idea of a shift "away from the sinoauricular node" is open to question. Before discussing the problem at hand and presenting further cases, I desire to add that the rarity of P-wave inversion in lead 1 is also illustrated by the work of Sampson and Caplan *(3)* who, in 60 successive patients with acute active rheumatic fever, found only 1 with P-wave inversion in lead 1.

The following case *(6)* is that of a 43-year-old man who had scarlet fever at the age of 6 years and was told at age of 11 that he had an enlarged heart. Physical examination revealed a moderate funnel chest with an aortic diastolic murmur. His blood pressure was 160/90. A diagnosis of rheumatic heart disease and rheumatic valvulitis, inactive, with aortic insufficiency was made. A recent ECG (fig. 7) revealed inverted P waves in leads 1 and 2 with a diphasic P wave in aV_L. The P-R interval was 0.16 seconds. The tracing revealed no significant change from a previous tracing (fig. 8) taken in 1947.

In the latter case the impulse is moving away from the right arm, primarily downward towards the left leg, slightly to the left, and slightly anteriorly. This again, it is believed, probably represented a shift in the pacemaker, probably associated with altered conductivity. Conductivity must have been altered at the sites of the old and the new pacemaker because the properties of the cardiac conduction system and cardiac musculature are closely interrelated and impulse formation, whether ectopic or not, is closely associated with conductivity.

The third case is a patient whose ECG is shown in figure 6. This 21-year-old student nurse had chorea at the age of 5 years but gave no history of rheumatism, rheumatic fever, or growing pains. Because of tachycardia and a sedimentation rate elevated to 20 mm., ECG's were made at the hospital where she worked. They were within normal limits. As a Navy dependent she reported to the outpatient department at this hospital for further study. She was afebrile and asymptomatic, offering no complaints whatsoever. Physical examination revealed a functional basal systolic murmur. No significant abnormalities were

(6) A patient of Dr. Albert C. Douglas, Consultant to U. S. Naval Hospital, St. Albans, N. Y.

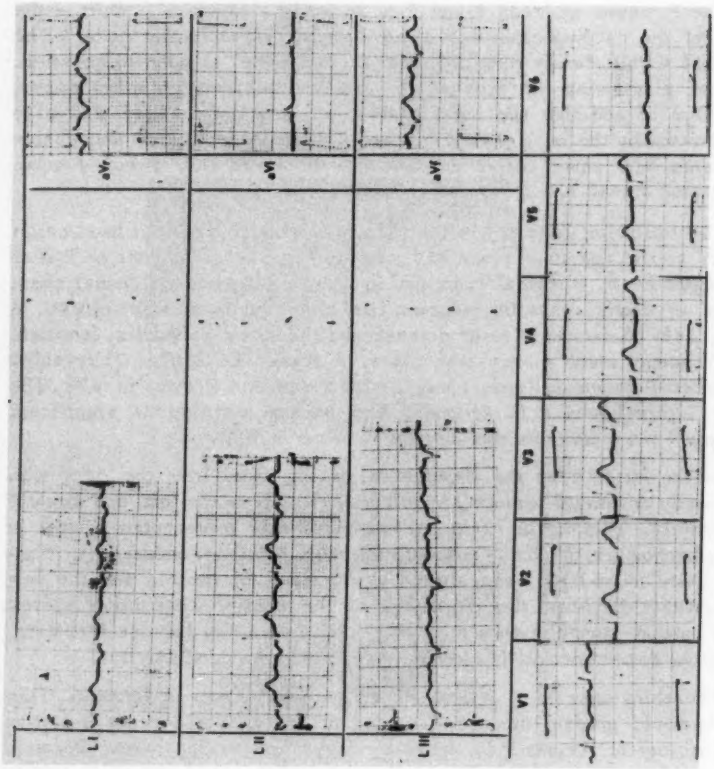

Figure 7.

found. The pulse varied between 80 and 90 beats per minute. The sedimentation rate was 10 by the Wintrobe method.

An ECG (fig. 9) appears to be similar to that shown in figure 1 in that the P waves are inverted in leads 1, 2, 3, and aV_F. They are diphasic in lead aV_L and upright in leads V_1 and V_2 (but not in lead V_3 as in figure 1). The P-R interval here is definitely lengthened to 0.22 seconds. Although possible, it seems unlikely that in the presence

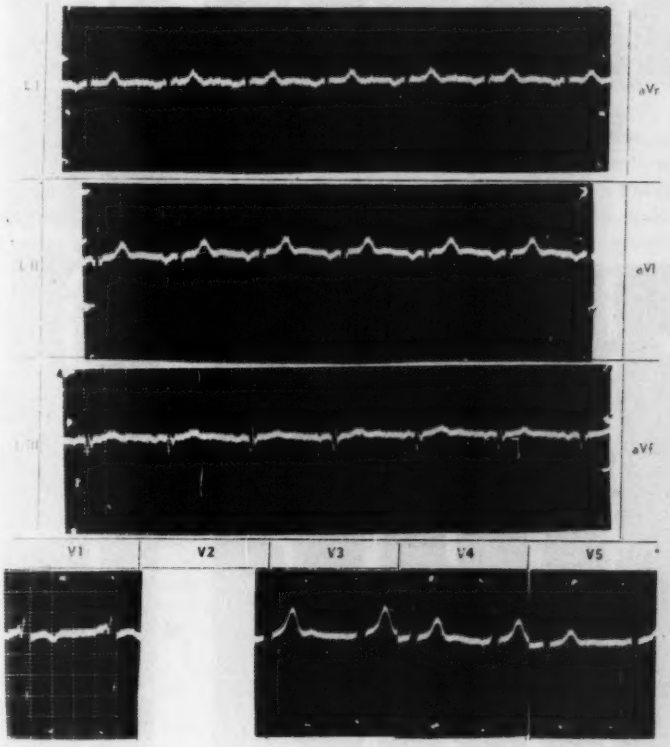

Figure 8.

of a first-degree heart block, again with P_1 inversion, the P-wave abnormalities are solely due to a high A. V. nodal rhythm. In the last 2 cases cited the abnormal form assumed by the P waves appears to be of significance insofar as the disease process, rheumatic fever, is concerned. Sampson and Caplan *(3)* in discussing aberrations of the P wave in acute rheumatic fever, including P-wave inversion, stated that: "These P wave deviations are regarded as diagnostically important in that they often occurred unaccompanied by other electrocardiographic abnormalities and occasionally during periods when

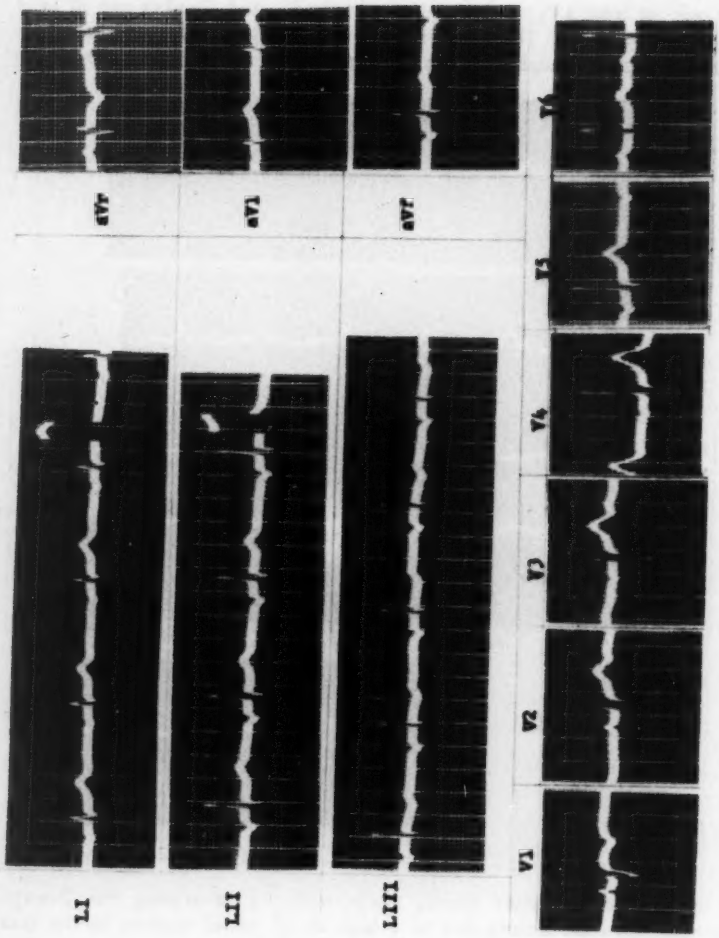

Figure 9.

clinical signs of active rheumatic fever were associated with normal temperature, leukocyte count, and erythrocyte sedimentation rate."

Such P wave changes may be produced by mechanisms other than a shifting of the pacemaker to the A. V. node. These mechanisms may possibly, at times, include some degree of myocardial damage which may be permanent inasmuch as these changes, as exemplified in case 2, would appear to remain static over many years.

SUMMARY

To date we have no definite explanation for the aberrations of the form of the P wave as pictured in the afore-mentioned 3 cases, but the time-accepted statement that P-wave inversion in leads 2, 3, and aV_F is necessarily indicative of a nodal rhythm is seriously questioned. In addition, 3 unusual and rather rare cases of inversion of the P wave in lead 1 are presented, 2 in rheumatic fever patients and 1 in a healthy, normal man. In the latter case, the P-wave inversion could be induced by right carotid sinus pressure and not by left carotid sinus pressure. The right carotid sinus, which acts primarily on the sinus node, is presumed to have shifted the pacemaker, probably to a low sinus rhythm rather than a high A. V. nodal rhythm.

BOOK REVIEWS

Introduction to Medical Science, by *Julius Jensen,* Ph. D., (In Medicine) University of Minnesota, M. R. C. S. (England), L. R. C. P. (London), Formerly Assistant Professor in Clinical Medicine, Washington University; Medical Service, St. Luke's Hospital; Staff Member, St. Louis City (Starkloff Memorial) Hospital and St. Louis County Hospital, and *Henry W. Noller,* M. D., Associate, St. Luke's Hospital; Physician, St. Luke's Hospital School of Nursing, St. Louis. 533 pages; illustrated. The C. B. Mosby Co., St. Louis, Mo., publisher, 1952. Price $5.75.

This new book will prove tremendously valuable to the student nurse, the physiotherapist, dietitian, and laboratory technician. It provides general medical information to those who could profit from an understanding of the problems of the physician without having to master the intricacies of the practice of medicine. The author (1) gives a concise but rather complete survey of the history of medicine, (2) presents a clear classification of diseases, (3) explains the doctor's approach to diagnosis and treatment of disease, (4) discusses the importance and help of the diagnostic laboratory, (5) emphasizes the great importance of preventive medicine, and (6) provides enough references to help the student in case more complete information is desired.—*Lt. Col. J. R. Vivas, MC, U. S. A.*

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Doctors in Blue, The Medical History of the Union Army in the Civil War, by *George Worthington Adams.* 253 pages; 16 illustrations. Henry Schuman, Inc., Publishers, New York, N. Y., 1952, Price $4.

This book was written as a doctoral thesis in history at Harvard. All phases of the American Civil War have been of perennial interest to the historian and the medical field is one that has been somewhat neglected in spite of the fact that the sources of information are very extensive. Dr. Adams, in a single volume, gives an account of the salient features of the medical and surgical aspects of the conflict. He deals with the confusion and difficulties in the expansion of the medical facilities of an army of 15,000 officers and men to a force to which nearly 2,500,000 were recruited during the 4 years of the war. He describes the work of Letterman and others in creating the ambulance system used along the lines originated by Napoleon's great surgeon, Larrey; the establishment of the general hospitals; the use of women nurses; and the many advances in administrative powers and methods of the Medical Department of the Army. The tremendous burdens carried by the Medical Department are realized when it is known that there were about 360,000 deaths from wounds and disease in the Union forces. At Gettysburg about 15,000 Union and 6,000 Confederate wounded were cared for after the 3-day battle. There are a few errors, as in referring to tartar emetic as a mercurial, but they are of minor importance in what is a well-written narrative. There are a number of well-chosen pictures and the format and printing of the book are excellent.—*Capt. L. H. Roddis, MC, U. S. N. ret.*

An Introduction to the Study of Viruses, by *Kenneth M. Smith,* F. R. S., Director of the Plant Virus Research Unit, Agricultural Research Council, Molteno Institute, Cambridge, Mass. 106 pages; illustrated. Sir Isaac Pitman & Sons, Ltd., London, England, publisher, 1950. Price $2.75.

Virus history dates from 1700 B. C. with the recognition of smallpox in China, but not until 1892 was a scientific demonstration of a virus made. Modes and probable modes of transmission are interestingly discussed with special reference to arthropods. Peculiarly enough, rickettsial diseases are included. Typical virus diseases of plants and animals are presented with laboratory technics for purification of the causative agents. Physical and chemical properties are given with electron microscopic studies and resolution of the virus into native virus protein which is free of nucleic acid and is noninfectious. The serology of viruses points up the importance of related strains and the use of immune serums in their identification. Some new points in the control of virus diseases, especially in plants, are given.

—*Col. R. C. Gaskill, MC, U. S. A.*

Gas Bacillus Infections

George R. Prout, *Lieutenant, junior grade, MC, U. S. N. R. (1)*

Robert B. Brown, *Captain, MC, U. S. N. (2)*

THE material for this report has been obtained from a patient load of about 9,000 casualties from the Korean campaign treated between 20 September 1950 and 4 August 1951. We have included only those patients with a spreading, virulent clostridial myositis. These are the cases of true gas gangrene and are not to be confused with infections produced by gas-forming organisms other than the clostridiums nor with clostridial cellulitis or localized myositis. Fifteen patients in this group presented the signs and symptoms of a spreading myositis. All underwent at least one surgical procedure at this facility. Three patients had definite evidence of major arterial damage; one had a transection of his brachial artery, one had his popliteal artery ligated, and one had suffered a crushing injury to his pelvis and right thigh.

In this last patient the right lower extremity was cold and pulseless on admission. Surgical exploration revealed a dissecting aneurysm of the right common iliac artery which was treated by ligation. Four days later it was necessary to do a midthigh amputation on the right because of vascular insufficiency. Eight days after the second procedure a progressive necrotic process had involved the stump to an extent requiring hip joint disarticulation. A demonstrable, widespread, vascular thrombosis in the stump may have initiated the gangrenous process, but the clinical and pathologic findings were compatible with a diagnosis of clostridial myositis of a nongaseous nature. Bacteriologic confirmation was not obtained.

There was 1 death in our series. Cole and Elman *(3)* mention that the mortality due to gas gangrene in World War I was 46.6 percent and that early in World War II MacLennan reported a 50 percent mortality for the British troops in the Middle East. The Seventh Army in 1945 reported a 24 percent mortality for casualties suffering gas gangrene in Europe. Although our series is so small that a death rate of 6.6 percent is not particularly significant, it encourages us to continue with our present mode of treatment.

(1) Formerly resident in surgery, Grasslands Hospitals, Valhalla, N. Y.

(2) Chief of Surgery, U. S. Naval Hospital in the U. S. S. *Repose.*

(3) Cole, W. H., and Elman, R.: Textbook of General Surgery. 5th edition. Appleton-Century-Crofts, Inc., New York, N. Y., 1948. p. 1092.

DIAGNOSIS

We have based our diagnosis on several factors. On the ward, the appearance of a toxic, unusually alert, apprehensive man with a fresh wound should excite suspicion. A rapid bounding pulse between 106 and 130 is associated with a temperature of 101° to 102° F. in most cases. The fetid, slightly sweet odor of the drainage from the wound (caused by saprophytes growing in symbiosis with the clostridiums) is virtually unmistakable. Crepitation early in the course of the disease is frequently not present and may lull the unwary into a sense of false security. Increasing pain in and about a wound is rarely absent if a clostridial myositis is present and should never be disregarded. We have not cultured wounds routinely for clostridiums but have cultured nearly all those in which gas gangrene was suspected or apparent. The results are shown in table 1.

TABLE 1. *Results of wound culture in series of 15 patients*

Organism	*Number of wounds*
Cl. perfringens only	8
Cl. perfringens predominating	4
No *Cl. perfringens*	1
Not cultured	2
Total	15

Altemeier and Furste *(4)* present widely divergent figures from those of various other authors with regard to the relative incidence of the different clostridial pathogens cultured from wounds. Our figures for *Clostridium perfrigens* were about 10 percent higher than the average for other authors listed. We made no attempt to culture, isolate, and identify the individual members of the group because the treatment is the same for infection with all pathogenic clostridiums, but a report of culture of *Clostridium septique* or *Clostridium oedematiens* may help to explain the lack of crepitation found at the bedside.

Roentgenograms of the injured area are frequently of assistance in making a diagnosis of gas gangrene, particularly if the wound is deep seated, the skin perforation is small, and the muscle involved is covered by strong, thick fascia. Eleven patients were examined roentgenographically. Gas was reported in 7. The small areas of decreased density scattered along the tract of a missile do not necessarily represent the products of a clostridial infection, but when the gas tends to produce an elongated pattern that follows fascial planes, it is virtually certain that some gas-forming organism is invading the tissues.

(4) Altemeier, W. A., and Furste, W. L.: Collective review; gas gangrene. Internat. Abst. Surg. 84: 507-523, 1947; in Surg., Gynec. & Obst. June 1947.

LOCATION OF INFECTIONS

The lower extremity is most frequently the site of gas gangrene, the thigh being involved most often. In our series 7 infections occurred in the leg, 6 in the thigh, 1 in the buttock, and 1 in the arm. One wound of the thigh resulted in retroperitoneal involvement and the only death.

TIME INTERVAL

The average time from wounding to recognition and treatment of the disease, excluding a patient with a crushing injury of the abdomen, was 2.5 days.

PREVIOUS TREATMENT

It is to the credit of the front-line surgeon that we had so few patients with gas gangrene when the gross contamination of the soil in Korea is considered. In the patients who did develop gas gangrene the following factors were observed: (1) extensive muscular damage; (2) major vascular injury associated with unevacuated, tense hematomas; (3) tight circular dressings; and (4) ill-fitted casts. Too frequently we received a report that a débridement had been performed on a given wound, but examination revealed excision of the skin and little else. Trueta (5) has said that no primary suture should be placed in any war wound from which muscle has been excised. It is our opinion that few war wounds should be sutured primarily. In at least 1 patient in this series sutures were used to close a wound of the thigh with the subsequent development of clostridial myositis.

CASE REPORTS

Case 1. A 21-year-old soldier was admitted to the U. S. Naval Hospital aboard the U. S. S. *Repose* on 12 October 1950 after he had sustained a wound of the left buttock 3 days earlier as the result of machine gun fire. He had received tetanus toxoid and penicillin (amounts unknown) and a débridement of the left buttock with transection of the glutei was performed before his arrival aboard ship.

On examination he appeared toxic but rational. His temperature was 101.2° F.; his pulse was 134; and his respiratory rate was 26. He complained bitterly of pain in the left buttock, numbness, and inability to move the left leg. A débrided wound of entrance was found over the crest of the left ilium and a wound of exit over the left buttock. Nonviable, wet, foul-smelling muscle protruded from the wound of exit and crepitation and tenderness extended over much of the buttock.

A diagnosis of gas bacillus infection was made and the patient was immediately skin-tested for sensitivity to gas gangrene antitoxin. Treatment with 500,000 units of penicillin every 2 hours was started.

(5) Trueta, J.: Principles and Practice of War Surgery With Reference to the Biological Method of the Treatment of War Wounds and Fractures. C. V. Mosby Co., St. Louis, Mo., 1943. p. 111.

He was given 43,000 units of gas gangrene antitoxin intravenously and 43,000 units intramuscularly after a negative skin test. A roentgenogram of the pelvis revealed a fracture of the left femoral neck and evidence of gas in the soft tissues. He was taken to the operating room within a few hours of his arrival. Under spinal anesthesia it was found that clostridial myositis had involved the entire left buttock. Extensive resection of all the diseased tissue included removal of the entire gluteal mass, the gemelli, obturator internus, and piriformis. The sciatic nerve was freed and left intact. After all visibly diseased tissue had been resected back to retractile, bleeding muscle, 200,000 units of penicillin in saline solution were instilled and a dry fluff gauze dressing over strips of petrolatum gauze was put in place. The patient received 1,000 cc. of whole blood during the procedure. The penicillin dosage was increased to 1,000,000 units every 3 hours and treatment with 250 mg. of aureomycin every 6 hours was started on the day of operation.

On the following day the patient's temperature fell to 100.2° F. but the radial pulse remained at 120. He was given 43,000 units of gas gangrene antitoxin intravenously and 43,000 units intramuscularly. The dressing of his surgical wound was changed and no evidence of persistence of the disease was noted. He continued to have temperature elevations as high as 102° F. until the fourth postoperative day when his fever subsided and did not return. His pulse rate fluctuated between 80 and 120 during this period but it returned to normal limits as the temperature dropped.

On the sixth postoperative day the penicillin was decreased to 50,000 units every 3 hours. The surgical wound had been dressed each day and no gross signs of infection were noted. On 20 October the area uncovered by skin was estimated at 6 by 10 inches and on 23 October (11 days after the radical excision) a secondary closure was performed using fine silk. The patient's immediate postoperative course was uneventful and on 25 October he was evacuated to Japan.

Case 2. A Marine sergeant sustained a large through-and-through wound of the right thigh as a result of a mortar burst. He was wounded 36 hours before his arrival at this activity. Previous treatment consisted of débridement of the wounds of entrance and exit and then partial closure by suture of the wound of exit. He had received 300,000 units of penicillin daily, 0.5 gram of streptomycin b. i. d., and 0.5 cc. of tetanus toxoid before admission. His only complaint was pain in the right thigh.

His temperature on admission was 101° F.; his pulse was 88; and his respiratory rate was 22. The wound of entrance, lying almost directly anterior in the midthigh, measured 3 by 5 inches. The wound of exit, measuring 4 by 6 inches, lay posterolaterally. Muscle and fascia protruded from both wounds but the tissue appeared viable. A mildly fetid odor was noted but no crepitation was palpable. Sutures had been placed

in the posterior wound of the thigh. The patient was given 500 mg. of aureomycin every 4 hours and 300,000 units of penicillin b. i. d. On the next day liquefaction necrosis in the muscle, subcutaneous crepitation, and changes in the skin color were noted. The pulse rate had risen to 108 and the temperature to 101.8° F. A diagnosis of gas bacillus infection was made. The patient was skin-tested and roentgenograms were made which revealed gas in the soft tissues of the injured thigh. He was given 43,000 units of gas gangrene antitoxin intravenously and 43,000 units intramuscularly and he was taken to the operating room.

Under spinal anesthesia the 2 wounds were connected on the lateral surface of the thigh and a large mass of vastus lateralis and vastus intermedius along with the rectus femoris was excised. In order to excise all infected muscle, it was necessary to expose about 3 inches of his femoral shaft. Fluffs were applied over thin strips of petrolatum gauze. He received 500 cc. of whole blood during the procedure and 500 cc. later on the ward. He continued to be mildly septic for the next 3 days, running elevations of temperature up to 101° F. with a pulse of 130 at times. He was given 600,000 units of penicillin every 6 hours and 0.5 gram of aureomycin every 4 hours. Treatment with sulfadiazine was started on the night of operation with an initial dose of 2 grams to be followed by 1 gram every 4 hours.

A secondary closure was performed early in the postoperative course. It was necessary to leave a palm-sized defect in the center. Local areas of infection occurred around some of the sutures but these responded well to treatment. About 2 weeks later a split-thickness graft was placed over the central defect and 5 days later it was evident that we had an 85 percent take. Two weeks after the graft the patient became fully ambulant and was evacuated to Japan 9 days later.

The 2 cases above were typical of the patient who has recently developed gas gangrene and has the usual complaints and findings associated with the disease. They also illustrate the type and amount of surgical treatment necessary to effect a cure. Secondary closure and skin grafting should be undertaken as soon as the operative sites fulfill the requirements necessary for those procedures.

Case 3. A 22-year-old soldier was admitted to this hospital on 2 October 1950 after sustaining a shell-fragment wound of the right thigh 2 days earlier. Shortly after he was wounded he began to experience severe abdominal pain, made worse by coughing. He had neither vomited nor experienced nausea. The only therapy he had received was 300,000 units of penicillin.

On admission his temperature was 101° F.; his pulse was 115; and his respiratory rate was 22. Rales were audible in the base of his chest on the right side. His abdomen was rigid, particularly in the right lower quadrant where exquisite pain accompanied palpation. Rebound tenderness was present. Brawny cellulitis was observed over his right

inguinal ligament. A small wound of exit lay just below this ligament. A large ragged wound of entrance was located 10 cm. below the ligament. A foul-smelling, dirty, thin discharge escaped from the wounds when the dressings were removed. Gas bubbles could be expressed from the wound on pressure.

A diagnosis of gas bacillus infection was made and the patient was skin-tested for sensitivity to gas gangrene antitoxin. Roentgenograms revealed large amounts of gas in the abdominal wall and thigh. He was given 43,000 units of gas gangrene antitoxin intravenously, 43,000 units intramuscularly, and 1,000,000 units of aqueous penicillin. It was obvious from the time of his arrival on the ward that he was dangerously ill and he was prepared for emergency operation.

Under pentothal, nitrous oxide, and oxygen anesthesia, an incision was made from just above the knee up to and across the inguinal ligament to the right flank. Clostridial myositis had involved half of the gracilis, nearly all of pectineus, and over half of the adductor mass and extended medially and laterally above the inguinal ligament. The involved portions of the above-mentioned muscles were excised to bleeding muscle and the femoral vein which had been perforated by one of the missiles was ligated in the femoral triangle. Above the inguinal ligament, the process extended medially down into the pelvis retroperitoneally. This space was drained with a large Penrose drain. Laterally, the inguinal canal was opened to the iliac spine. The retroperitoneal space there was opened and foul, serosanguineous fluid escaped. Two large foreign bodies were removed from the iliopsoas muscle. The peritoneal cavity was opened purposely and when found clean was immediately closed. The lateral retroperitoneal space was also drained and a loose dressing covered by a spica was applied to the thigh.

The patient went into peripheral vascular collapse 10 minutes after the induction of anesthesia and remained in this state throughout much of the ensuing procedure. He received 2,500 cc. of whole blood during the operation and was sent to the ward in a critical condition. He died about 4 hours later in spite of supportive measures. Autopsy revealed cardiac failure, pulmonary edema, hepatic necrosis, and nephrosis.

This patient was brought aboard in desperate condition. It is probable that because of involvement by clostridial myositis of muscle which could not be reached no form of therapy would have saved him. If surgical excision cannot be performed, the prognosis is poor regardless of the type and amount of general supportive therapy rendered.

TREATMENT

Our treatment has been based on 2 specific principles as illustrated above.

1. The general care of the patient must commence immediately. After obtaining a negative skin test, he should receive 4 ampules of a trivalent gas gangrene antitoxin, each ampule containing 10,000 units of *Cl. perfringens,* 10,000 units of *Cl. septique,* and 1,500 units of *Cl. oedematiens* antitoxin. It has been our practice to give 2 ampules intravenously and 2 intramuscularly as soon as the diagnosis is made. This dosage, or half of it, may be repeated in 6 hours and as the indication arises. At least 1,000 cc. of whole blood should be made available immediately and the patient taken to the operating room where the second phase commences. We have given at least 2.4 million units of penicillin per day in divided doses as 2-, 3-, or 6-hour intervals and this seems to have been sufficient. In addition, we have given sufficient sulfadiazine orally to reach a concentration in the blood of from 12 to 15 mg. per 100 cc. The usual precautions were observed when this drug was used. Aureomycin was given on a dosage schedule of 0.5 gram every 6 hours.

2. The surgical operation must be designed to remove all nonviable muscle. If a muscle is faintly discolored but twitches on stimulation and bleeds readily, it may be left; but if it is noncontractile or avascular, it must be removed.

Extensive debridement was accomplished in 11 of our patients. The diagnosis of gas gangrene was made on 1 patient in a forward area and he underwent a below-the-knee amputation there. On arrival here we had to redissect the stump because of persistence of the disease. It was necessary for us to perform 4 amputations: 1 midarm, 2 supracondylar in the thigh, and 1 below the knee. All were of the guillotine type.

CONCLUSIONS

In a series of 15 patients with clostridial myositis one who was in a critical condition on admission died. Although this series is small, the following observations may be made:

1. The earlier the diagnosis is made, the better the chance to save life and limb. An exploration resulting in negative findings is to be preferred to procrastination.

2. Surgeons in the forward echelons should perform as complete a débridement as possible, avoid applying tight or ill-fitting casts, evacuate hematomas, and control hemorrhage. They should not attempt the primary closure of war wounds.

3. We believe that immediate operation, trivalent gas gangrene antitoxin, large doses of penicillin, sulfadiazine, and aureomycin are indicated in treatment. Large transfusions of blood are frequently needed. The surgical procedure should be extensive with consideration to function and anatomy and all involved muscle should be removed.

BOOK REVIEW

Wounds of the Extremities in Military Surgery, by *Oscar P. Hampton, Jr.*, M. D., F. A. C. S., Instructor in Clinical Orthopedic Surgery, Washington University School of Medicine; Attending Orthopedic Surgeon, Barnes Hospital, St. Louis City Hospital, St. Louis County Hospital, St. Luke's Hospital; Colonel, U. S. Army Reserve; Civilian Consultant to the Surgeon General, Department of the Army; Chief, Orthopedic Section, 21st General Hospital, 1942-1943; Consultant in Orthopedic Surgery, Mediterranean Theater of Operations, 1943-1945; Chief, Orthopedic Branch, Surgical Consultants Division, Office of Surgeon General, U. S. Army, 1946. 434 pages; 131 illustrations. The C. V. Mosby Co., St. Louis, Mo., publisher, 1951. Price $10.

In several respects this book is unique. In the first place, the information is based on the most massive experience with traumatic wounds of the extremities in medical history, namely the Mediterranean (earlier the North African) Theater of Operations, in which the American Ground Forces were in combat almost continuously from 7 November 1942 until 2 May 1945. In the second place, the surgery of trauma (or military surgery) is treated in most books under the various surgical specialties so that no comprehensive picture is fixed in the mind. Such is definitely not the case in this work, where all phases (osseous, vascular, nervous, and muscular) of wounds of the extremities are considered, and the total treatment is outlined. Third, the author recognizes that no matter how well trained a surgeon may be, he is to a certain extent entering into a new branch of surgery when he is called on to treat battle casualties. Although basic surgical principles remain the same, the exigencies of military surgery, particularly the variable facilities available at various echelons, and the necessity for evacuation, make marked differences in his decisions and choice of procedures.

To the best of my knowledge, this is the only reference book of its kind in which the civilian surgeon who is about to become or who has just become a military surgeon can find how to integrate the exigencies of military surgery with his previous professional experience. In addition, however, the civilian physician who treats traumatic injuries will also find it a valuable reference text because of the broadness of its basic surgical principles.—*Lt. Col. P. A. Reed, MC, U. S. A.*

Psychotherapy in a Recruit Training Center

Arthur H. Chapman, *Lieutenant, USPHS(R)* (1)

CONDUCTING a psychotherapeutic service in a boot camp involves certain problems of technic peculiar to the situation. The factors which tend to make the situation unique are:

1. The total period of basic training for the recruit is 12 weeks. Because the patient coming for psychiatric help will come after having been in camp an average of at least 2 weeks the time for effectively working with him is further reduced.

2. The usual supporting and familiar figures in the patient's life (the parents, siblings, and others) are not present. This greatly restricts the availability of any collaborative anamnestic data or evaluation of the patient's personality development by some direct contact with those persons who have been most important in the emotional milieu of the patient's upbringing.

3. Environmental change by the patient through insight or by the psychiatrist through direction is virtually impossible without a gross interruption in the recruit's military training.

4. There is a large gap in military rank between the recruit and the psychiatrist. The establishment of rapport is made more difficult by this artificially interpolated factor in the interview relationship.

5. By virtue of the military situation the psychiatrist is a much more authoritative figure in the patient's eyes than in an equivalent civilian treatment situation. This is a factor which not only tends to inhibit the patient, but also tends subtly to corrupt the countertransference of the psychiatrist in megalomanic and omnipotent directions.

6. The immediate value of secondary gains from neurotic illness to avoid rigorous situations in duty is much greater than in civil life. Successful secondary gains from neurotic illness in this situation not only are not in the true interests of the patient, but are also a detriment to the morale of the other recruits with whom the patient associates.

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These factors require that technics of psychotherapy fall within the following circumstances if the psychotherapy is to be consistent with the realities of the boot camp situations:

1. The technics must be aimed at accomplishing the maximum within a few weeks.

2. The patient's main problems must be evaluated rapidly and dealt with directly if anything more than palliative work is to be done.

3. The patient must quickly adjust to the situation he is in because the military situation does not allow any appreciable change or reorganization of the base or its training program to meet the personality structures of individual recruits.

4. The rapport barrier created by the difference in rank of patient and therapist must be universally assumed and dealt with quickly and briefly. Extensive time for breaking down rapport barriers is not available.

5. The therapist must be aware of the possibilities of megalomanic countertransference feelings toward the patient. His vigilance must be constant and his self-examination critical in this respect.

6. The psychiatrist must avoid opening the floodgates of secondary gains insofar as possible while being scrupulous about the best interests of the patient and never underestimating or underevaluating his symptoms.

I believe that an effective psychotherapy is practicable despite the limitations referred to. An evaluation of how effective the psychotherapy is working can be much more rapidly gained in a recruit training center than in a civilian situation. In the camp the patient is literally under continuous surveillance by the training staff. The psychiatrist can receive accurate reports on the behavior, emotional ease, and adjustment of the recruit from those who are in close contact with him. This differs from civilian practice where the psychiatrist must often speculate on the realities of the patient's daily adjustment and often much time must elapse before the patient's defenses are sufficiently penetrated that an accurate evaluation can be made. The "demerit" system commonly used in boot camps offers an auxiliary objective impression of adjustment to the requirements of service. Consequently, impressions as to how effective the psychotherapeutic technics are become fairly evident after a period of a few months. Such an evaluation implicitly centers on the adjustment of the recruit to the military situation and symptomatic relief and greater emotional comfort of the patient in boot camp. Regarding the long-range better adjustment of the patient, the camp psychiatrist can form only tentative impressions. Such impressions are worth noting but have no assured validity. It is the general impression that a successful solution of problems in boot camp and progress there on emotional difficulties definitely gives the recruit finishing training a legacy of greater mental health and adaptability to carry into other service assignments and later civilian situations.

There is a tendency for many boot camp problems to fall into patterns which are seen repeatedly from case to case. Though the details may be different for different patients, it is striking how similar the basic problems of many recruits are. This would seem to stem, at least partially, from the fact that the recruits are all required to meet a standardized new life situation under similar circumstances. It is therefore not surprising that a large percent of the patients seen would tend to fall into similar categories. Moreover, these similar problems seem to lend themselves to similar psychotherapeutic measures.

The patterns of 4 of the commonly seen problems are as follows:

1. The *subnormally-assertive, passive* recruit who inhibits all expression of hostility and assertiveness usually shows anxiety, ranging from acute anxiety states to tearful panic states. Technics are designed to give the recruit insight into how he handles his hostilities and the resultant production of symptoms. He is then urged to adopt acceptable and moderate verbal outlets for these emotions similar to those used by his barrack mates. His ability successfully to adopt such modes of behavior with ease seems distinctly greater than that of similar patients in civilian practice. The recruit in acute anxiety, robbed of the usual parental refuge and isolated from his usual environmental supports, forms a strong and rapid identification with the psychiatrist who stands as his main support and plays multiple parental roles simultaneously. Through the strength of this identification with the therapist the recruit has a marked ability to incorporate behavior patterns suggested by the psychiatrist. The rapid and striking identification which the recruit makes in therapy seems to enable him to make much faster progress in this area of expression.

2. The recruit with *strong dependency needs* who requires a buffer for weaning himself away from the home situation undergoes brief periods of depression and tearfulness, often with a variety of physical complaints. A sympathetic interpretation of his feelings and symptoms in terms of ruptured dependency-gratifying home situations, plus establishment of a brief term dependency-need buffer in the person of the psychiatrist, is quite effective. The process of resolving his anxieties about his dependency needs can be made a constructive step in his personality maturation. He can be given a great deal of relief with relatively little expenditure of time. The interviews, after the first one, can be very brief yet effective.

3. The *isolated, withdrawn, schizoid* recruit who needs to develop better technics and comfort in interpersonal relationships is usually referred by the training staff with the statement that "the boy just isn't fitting in and he doesn't mix with the other men." To these recruits, service can be presented as an opportunity for developing new ways of meeting and interacting with people which will be not only an asset in the service, but also in civil life later. Socializing technics are discussed and urged. Such psychotherapeutic measures with isolated,

schizoid persons in civil life are largely futile, but the results from such measures with recruits are often gratifying. The only explanation for this difference would seem to be the rapid and firm identification of the recruit with the psychiatrist and his ability to incorporate successfully many of the attributes inherent in his concept of the therapist.

4. The recruit who feels *guilt over leaving home,* having volunteered for military service as a means of escaping from a difficult home situation, may become very anxious about his parents whose attitude was obviously very demanding, rejecting, and depreciating toward him. The psychotherapy here must be much more flexible. As a rule much relief is afforded by allowing the recruit a simple catharsis of his emotions and making interpretations for him of the nature of his guilt feelings. An objective, verbalized examination of the realities of the home situation will generally suffice. Analysis of the ways in which the parents have manipulated his guilt feelings and anxieties in the past in order to dominate him is far too much for the brief period available for psychotherapy.

These are the most common patterns of emotional problems met in a boot camp. They constitute less than half the cases seen, but are sufficiently common and repetitive in pattern to merit special discussion.

The psychotherapy of other recruit problems has been most effective and most in keeping with the realities of the situation when the following general formulations of technic have been followed:

1. Medication, as an auxiliary aid, has been very disappointing. The recruit often seems disappointed when a medication is suggested, although this has always been done with the explanation that the medication would play a secondary role and was designed only to give a measure of relief while the basic personality difficulties were being treated. The recruit seems to feel that an offer of medication is a betrayal of his pressing dependency needs and looks on medication as a rejection. The usefulness of medication in civilian psychotherapy is also quite limited. Its usefulness in treating recruits is even more limited and constitutes a liability more often than an asset. The recruit can tolerate his anxiety and depression symptoms much more easily than he can tolerate any suggestion of a "brush-off" by the psychiatrist.

2. Because of the marked brevity of time allowed for psychotherapy, dynamics must often be speculatively and tentatively inferred from the material at hand. Interpretations must be made much sooner and much more directly than when a more extensive and leisurely time for psychotherapy is available. To avoid rejection by the patient of this rapid interpretation technic, I use "interpretation in a tentative sense." In this, the therapist offers his interpretation frankly as a general impression he has gained from the material, to be considered by the patient and verified or not as further material comes out. In this way the dangers of premature interpretations are largely mitigated. Hence, the in-

terpretations, made from the therapist's insights, often precede the material that verifies it, as opposed to the technic used when more time is available. The interpretation is frankly and sincerely given as a tentative impression and no attempt is made to sell it to the patient. Little difficulty is encountered in the relatively rapid assimilation of interpretations when they are given in this manner.

3. Interpretations that would mobilize new guilt or new anxiety are not given. The brevity of time allows only the handling of the areas in which anxiety and guilt, and their ineffective symptom defenses, are already mobilized. Exploration of areas where new anxieties or guilt may be mobilized is not broached. There is, as a rule, no assurance that there would be sufficient time to handle these areas if they were to be opened.

4. Therapy is often best approached with the patient as a growth process in which therapy opens up new vistas of insight and removes anxiety-ridden blocks to more effective and comfortable interpersonal relationships. Thus therapy can be conceived by the patient as one step in a total process of adjustment to service and to life. It can be conceived as a continuous process with no proper end point, but a process which, once begun by therapy in boot camp, will continue to have a certain autonomous vitality and progression of its own. The usefulness of this metaphorical and pictorial concept of therapy with the recruit is marked. It gives therapy a cohesive pictorial concept useful to the nature of therapy and the time limitations involved.

5. Transference is more exploited than interpreted. A positive transference is tacitly encouraged by the therapist's friendly and interested demeanor. This positive transference is a very helpful step to a ready identification with the therapist, and the incorporation of the therapist's interpretations and their translation into changes in behavior. When negative transference becomes apparent, it is good-naturedly interpreted and recognized. It may be interpreted in terms of past hostilities against authoritative figures or in terms of current hostilities to service authorities. Among new recruits, hostility to service authorities is rarely a persistent block in therapy. Any negative transference must be quickly dealt with as soon as evidence of it appears because time for its extensive analysis is not available.

The results of psychotherapy with recruits are gratifying. The recruits are in a young age group and are easily adaptable; they are, moreover, away from their original symptom-producing environments which in civilian practice often sabotage the painstaking work of the psychiatrist.

BOOK REVIEW

A Textbook of Orthopedics, With a Section on Neurology in Orthopedics, by *M. Beckett Howorth,* M. D., Clinical Professor of Orthopedic Surgery, New York University Post-Graduate Medical School; Formerly Assistant Clinical Professor of Orthopedic Surgery, College of Physicians and Surgeons, Columbia University; Associate Attending Surgeon, New York Orthopedic Hospital; in association with *Fritz J. Cramer,* M. D., *A. Wilbur Duryee,* M. D., *Donovan J. McCune,* M. D., *J. William Littler,* M. D., and *Walter A. Thompson,* M. D. 1,110 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publishers, 1952.

In the author's words, "The purpose of this book is to provide the basic facts of the etiology, pathology, diagnosis, and treatment of abnormalities and diseases of the musculoskeletal system for the physician interested in these problems." It is a comprehensive text, embracing the entire field of orthopedic surgery. For this reason it should be enthusiastically received as most of the works on orthopedics published in English in the last decade have been limited to a single phase of the subject.

The author divides his text into anatomic and etiologic classifications, perhaps risking some repetition, but on the credit side this allows the student to correlate the variety of orthopedic conditions affecting a region. The last division, "Neurology in Relation to Orthopedic Practice," by an associate author, is particularly valuable for the student. The volume is profusely illustrated with photographs, charts, diagrams, and roentgenograms which are excellently chosen and skillfully reproduced. Historical and contemporary references follow each section and are numerous, comprehensive, and up to date. The author is to be commended on producing a much-needed general text and on his readable style. The volume can be recommended to orthopedists, other surgical specialists, and to the general practitioner, as well as to students in the field of orthopedics and general surgery.

—*Commander W. C. Roland, MC, U. S. N.*

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